

**Biopsychological Investigation of Hedonic Processes in Individuals  
Susceptible to Overeating: Role of Liking and Wanting in Trait  
Binge Eating**

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The candidate confirms that the work submitted is her own, except where work which has formed part of jointly authored publications has been included. The contribution of the candidate and the other authors to this work has been explicitly indicated below. The candidate confirms that appropriate credit has been given within the thesis where reference has been made to the work of others.

Chapter 2 of this thesis was based in part on the jointly-authored publication:

Dalton, M., King, N.A., & Finlayson, G., (2013) Appetite, Satiety and Food Reward in Obese Subjects: A Phenotypic Approach, *Current Nutrition Reports*, 1-9.

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The candidate confirms that her contribution was primarily intellectual and she took a primary role in the production of the substance and writing of each of the above. Her co-authors confirm that their contribution to each of the articles was in guiding the research presented and its evaluation as well as editing drafts of the manuscripts.

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## Abstract

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The objective of this thesis was to identify and characterise a robust phenotype susceptible to reward-driven overeating. Specifically, the thesis aimed to examine the role of liking and wanting for food in trait binge eating and to determine other potential biopsychological markers of susceptibility (psychological, physiological and genetic).

In a systematic series of studies, normal-weight (Ch.6,7,9) and overweight or obese (Ch.7,8) females were categorised as either ‘binge-type’ or ‘non-binge type’ based on their scores on the Binge Eating Scale. Using a biopsychological approach, susceptibility was characterised across several different scientific domains. Liking and wanting for food were measured using the validated Leeds Food Preference Questionnaire (Ch.5-9) – LFPQ - which separated explicit and implicit processes. Food choice and energy intake were assessed objectively and quantitatively in the laboratory using ad libitum test meals (Ch.5-9) and under free-living conditions using a validated multiple-pass 24-hour dietary recall (Ch.8). Physiological markers (fat mass, fat-free mass) were measured using bioelectrical impedance and air plethysmography (Ch.6-9). Potential genetic markers of susceptibility (e.g. FTO, DRD2, Taq1A, CD36) and intermediary phenotypes of trait binge eating were examined using a candidate gene approach (Ch.9).

Overweight-obese ‘binge-types’ had enhanced explicit liking for food overall, and greater implicit wanting for high-fat sweet foods compared to overweight-obese ‘non-binge types’. This was associated with an increased preference for, and consumption of these foods under laboratory and free-living conditions. Furthermore, obese ‘binge-types’ had greater levels of adiposity and reported greater food cravings and lower positive affect. Lean ‘binge-types’ had a greater implicit wanting for sweet foods, and exhibited a greater preference for these foods. Liking

and wanting for food assessed by the LFPQ were related to energy intake and food choice. Notably, an enhanced liking for food in a fed state was associated with greater energy intake. In addition, implicit wanting emerged as an important process; while enhanced implicit wanting for sweet foods was a risk factor for overeating, greater implicit wanting for low-fat savoury foods appeared to be protective. Examination of the intermediary phenotypes revealed that variation in certain genes relating to reward, taste and obesity were associated with energy intake and food choice, body composition and food hedonics.

This thesis has identified a distinct, ecologically valid, behavioural phenotype of obesity that is characterised by reliable psychological and physiological characteristics. Furthermore, the results confirm the value of distinguishing between liking and wanting for food and for studying their role in eating behaviour.

## Publications and presentations

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### Publications

#### *Research Articles*

**Dalton, M.**, Blundell, J. & Finlayson, G. (2013) Examination of obese binge-eating subtypes on reward, food choice and energy intake under laboratory and free-living conditions. *Frontiers in Psychology*, **4**, 757.

**Dalton, M.**, Blundell, J. & Finlayson, G. (2013) Effect of BMI and binge eating on food reward and energy intake: further evidence for a binge eating subtype of obesity. *Obesity Facts*, **6**; 348-359

Finlayson, G., Arlotti, A., **Dalton, M.**, King, N.A. & Blundell, J.E. (2011). Implicit wanting and explicit liking are markers for trait binge eating. A susceptible phenotype for overeating. *Appetite*, **57**; 722-728

#### *Reviews*

**Dalton, M.**, King, N.A., & Finlayson, G., (2013) Appetite, Satiety and Food Reward in Obese Subjects: A Phenotypic Approach, *Current Nutrition Reports*, 1-9

Cecil, J.E., **Dalton, M.**, Finlayson, G., Blundell, J., Hetherington, M. & Palmer, C. (2012) Obesity and eating behaviour in children and adolescents: contribution of common gene polymorphisms. *International review of Psychiatry*, **24 (3)**; 200-210

Finlayson, G. & **Dalton, M.** (2012) Hedonics of Food Consumption: Are Food 'Liking' and 'Wanting' viable targets for appetite control in the Obese? *Current Obesity Reports*, **1**; 42-49.

Finlayson, G. & **Dalton, M.** (2012) Current progress in the assessment of 'liking' vs. 'wanting' for food in human appetite. Comment on "You Say it's Liking, I Say it's Wanting..." On the difficulty of disentangling food reward in man' *Appetite*, **58 (1)**, 373-378

#### *Book chapters*

**Dalton, M.** & Finlayson, G. (Oct 2013) Hedonics, satiation and satiety. In *Satiation, Satiety and the Control of Food Intake*

Blundell, J., **Dalton, M.** & Finlayson, G. (2013) Appetite and Satiety – A Psychobiological Approach. In Murcott, A., Belasco, W. & Jackson, P. *The Handbook of Food Research*. Bloomsbury Academic

Finlayson, G., **Dalton, M.** & Blundell J. (2012) Liking vs. Wanting Food in Human Appetite: Relation to Craving, Overconsumption and "Food Addiction". In Brownell, K.D. & Gold, M.S. *Food and Addiction: A Comprehensive Handbook*. Oxford University Press.

## **Oral and poster presentations**

### ***Oral presentations***

Is trait binge eating a hedonic subtype of obesity? 48hr free-living and laboratory-based examination of reward, food selection and energy intake. Paper presented at *Society for the Study of Ingestive Behavior*, New Orleans, USA, July 2013

Characterising trait binge eating through intensive 48 hour assay of food hedonics, macronutrient intake, food choice and overconsumption. Paper presented at *European Congress on Obesity*, Liverpool, UK, May 2013

Dissociation of liking and wanting for high-fat sweet foods characterise a binge-eating phenotype of obesity. Paper presented at *Canadian Obesity Student Meeting*, Edmonton, Canada, June 2012

Implicit wanting is a marker of a susceptible phenotype for overconsumption. Paper presented at *BBSRC DRINC 6<sup>th</sup> Dissemination Event*, Bristol, UK, April 2011

Individual differences in the hedonic control of overconsumption. Paper presented at *Institute of Psychological Sciences Research Seminar*, University of Leeds, UK, Feb 2011

### ***Poster presentations***

Preliminary evidence for the association of two common variants in the CD36 gene and reduced body fat in adults. Poster presented at *European Congress on Obesity*, Liverpool, UK, May 2013

Dissociation of liking and wanting for high-fat sweet foods characterise a binge-eating subtype of obesity. Poster presented at *Treatment of Obesity Conference*, Birmingham, UK, June 2012

Trait binge eating is a hedonic phenotype of obesity: Joint action of ‘liking’ and ‘wanting’ for food. Poster presented at *European Congress on Obesity*, Lyon, France, May 2012

Enhanced Liking and Wanting for Sweet Foods Characterise a Binge Eating Phenotype of Obesity. Poster presented at *BBSRC DRINC 7<sup>th</sup> Dissemination Event*, Manchester, Oct 2011

Implicit wanting is a marker of trait binge eating – a susceptible phenotype for overeating. Poster presented at *BBSRC DRINC 6<sup>th</sup> Dissemination Event*, Bristol, UK, April 2011

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## List of abbreviations

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AMPM: Automated Multiple Pass Method	L-B: Lean binge-type
ANCOVA: Analysis of covariance	LFPQ: Leeds food preference questionnaire
ANKK1: Ankyrin repeat and kinase domain containing 1	LFSA: Low fat savoury
ANOVA: Analysis of variance	LFSW: Low fat sweet
BAS: Behavioural Activation Scale	L-NB: Lean non-binge type
BBSRC: Biotechnology and Biosciences Research Council	MC4R: Melanocortin 4 receptor
BES: Binge Eating Scale	mm: Millimetres
BIA: Bioelectrical Impedance Analysis	ms: Milliseconds
BMI: Body mass index	O-B: Obese binge-type
CD36: Cluster of differentiation 36	O-NB: Obese non-binge type
cm: Centimetres	OPRM1: opioid mu receptor 1
COEQ: Control of Eating Questionnaire	PCA: Principal Components Analysis
DER: Daily energy requirements	PCR: Polymerase chain reaction
DRD2: dopamine receptor D2	PET: Positron emission topography
DR-EI: Dietary recall energy intake	SLC2A2: Solute carrier family 2 member 2
D-RT: Standardised d score	SNPs: Single nucleotide polymorphism
EEE: Estimated energy expenditure	SPSS: Statistical Package for the Social Sciences
EI: Energy intake	TAS1R2: Taste receptor type 1 member 2
fMRI: Functional magnetic resonance imaging	TAS1R3: Taste receptor type 1 member 3
FTO: Fat mass and obesity associated gene	TAS2R38: Taste receptor type 2 member 38
HARU: Human Appetite Research Unit	TFEQ: Three Factor Eating Questionnaire
HFSA: High fat savoury	TM-EI: Test meal energy intake
HFSW: High fat sweet	VAS: Visual analogue scale
Kcal: Calories	VPT: Visual Probe Task
kg: Kilograms	YFAS: Yale Food Addiction Scale

# Chapter 1

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## General Introduction

### 1.1 Obesity: Recent trends in the UK

The increased prevalence of overweight and obesity, while once limited to high-income countries, now presents a global concern (Finucane et al., 2011). In the UK, the latest statistics state that between 1993 and 2011, the number of individuals who were within the normal BMI range decreased from 41% to 33.6% for males and from 49.5% to 39.4% for females, and as of 2011 24% of males and 26% of females were classified as obese (Health Survey for England, 2011). This upward trend in overweight and obesity has been accompanied by considerable health and economic costs.

There are several diseases and health problems that are associated with weight gain and excess body fat, including hypertension, infertility, osteoarthritis, type 2 diabetes, heart disease and certain forms of cancer (Kopelman, 2007). Indeed, when both BMI and waist circumference are taken into consideration, it is estimated that of obese men, 18% are at increased risk, 15% are at high risk and 21% are at very high risk of obesity associated health problems. For obese women the figures are 15%, 18% and 26%, respectively (Health Survey for England, 2011). The many chronic and acute health problems associated with excess body weight not only negatively impact the individual with regards to reduced quality of life but also places a burden on society, as the cost of healthcare resources in the UK that are devoted to the treatment of overweight and obesity was estimated to be £4.2 billion in 2007 and is projected to rise to as much as £27 billion per year by 2015 (Butland & Britain, 2007). These statistics suggest that there is a need for the development of more effective treatment and prevention strategies for obesity, and a greater understanding of the contribution and likely interaction of the numerous causal factors is needed.

## **1.2 Causes of weight gain and obesity**

Weight gain and obesity occur when an energy imbalance is created in which energy intake exceeds energy expenditure over a prolonged period of time. However, this energy imbalance explanation assumes a simple relationship between energy that is taken in and energy that is expended. It does not take into account the complex set of interactions that arise from a range of different factors, including genetic, social and environmental factors, which ultimately contribute to the end result.

It is predominantly believed that changes in the environment are largely accountable for the current obesity epidemic. These changes include an increase in availability of foods that are highly palatable, energy dense and relatively inexpensive (Swinburn et al., 2011; Wadden, Brownell, & Foster, 2002) and a decrease in the energy cost of everyday life (Church et al., 2011). To this end, becoming overweight or obese has been described as a ‘normal response’, in which the homeostatic regulation of appetite and energy balance is challenged by environmental pressures to over-consume, with energy intake no longer primarily being driven by energy need but rather the rewarding aspects of food (Swinburn et al., 2011). Indeed, a fundamental imbalance in the homeostatic control of appetite is that while there are strong defence mechanisms in place to protect against substantial loss of body weight and fat mass, the mechanisms in place that mitigate long term increases in body weight and adiposity are comparatively weak (Erlanson-Albertsson, 2005). Therefore, both homeostatic and hedonic processes determine appetite and energy balance.

## **1.3 Homeostatic and hedonic systems of appetite control**

A sustained state of positive or negative energy balance depends on both *what* and *how much* food is consumed in relation to energy expenditure. The qualitative aspects of eating behaviour (what to eat) depend, at least in part, on the direction of food preferences, driven by the expectation and experience of pleasure obtained from food (wanting and liking for food). The quantitative aspects of eating behaviour (how much to eat) reflect a general drive, and inhibition of this drive, to eat (the

strength and duration of satiation and satiety). This distinction between drive and direction is often framed in terms of homeostatic and hedonic systems for the control of food intake (Blundell & Finlayson, 2008).

The homeostatic system refers to the regulation of food intake that arises from biological need and acts to maintain the internal environment and energy stores. It comprises a feedback network of hunger and satiety signals that influence the initiation and termination of eating (Berthoud & Morrison, 2008). Hunger peptides are released before a meal is initiated and include neuropeptide Y and ghrelin. Satiety signals are released in response to the ingestion of food, with some originating from the digestive tract, for example cholecystikinin (CCK) and glucagon-like peptide 1, and others being produced in the adipose tissue, for example leptin (Schwartz, Woods, Porte, Seeley, & Baskin, 2000). Signals of satiety act to bring about the termination of an eating episode, however, these signals act as a suggestion rather than an order and the rewarding aspects of food are able override or modulate signals of satiety. The hedonic system of appetite control refers to the sensory and external stimulation of food intake and takes into consideration that eating behaviour can be motivated by external cues in the environment and does not solely arise in response to energy need. Behavioural neuroscience studies have demonstrated that the hedonic system of appetite control appears to be underpinned primarily by opioid and dopamine neurotransmission (other neuro-chemicals have been implicated), with the opioid system mediating the hedonic impact or the degree of pleasure (liking) derived from food, and the dopamine system mediating the motivation (wanting) to obtain it (Berridge & Robinson, 2003). In studies of human appetite, liking and wanting for food are often viewed in relation to subjective states or explicit feelings that refer to the everyday understanding of these terms in the context of food choice and food intake (Finlayson & Dalton, 2012b). Wanting may describe subjective states of desire or craving, whereas liking is typically defined as the perceived hedonic impact of a food, or the appreciation of its sensory properties.



Liking and wanting as psychological components of reward are thought to operate at implicit (unconscious, automatic) and explicit (conscious, introspective) levels and may bear some relation to dual process models of motivation (e.g. Frieze, Hofmann, & Wänke, 2008; Wilson, Lindsey, & Schooler, 2000). Previous research has demonstrated that the homeostatic and hedonic systems of appetite control are underpinned by separate substrates and can be dissociated. For example, Yeomans and Wright (1991) administered an opioid antagonist, nalmeferne, or a placebo to participants before they tasted and rated the palatability of a number of different food items. They found that palatability ratings were significantly lower in the nalmeferne condition compared to the placebo condition. However, there were no differences in ratings of hunger between the two conditions. Conversely, in a sample of obese individuals, pharmacological suppression of hunger by the serotonin drug d-fenfluramine had no impact on the appreciation of the pleasantness of food (Blundell & Hill, 1987).

In addition to the evidence supporting dissociation between the two systems, research also suggests that there are interactions between liking and wanting with hunger and satiety. For example, research has demonstrated that increased pleasantness or liking of food is able to increase energy intake by increasing hunger and therefore delaying satiation. Yeomans, Gray, Mitchell, and True (1997) examined ratings of palatability and hunger during the consumption of either a bland or a palatable meal. They found that in the palatable food condition ratings of hunger sharply increased in the early stages of consumption and declined at a slower rate throughout the meal compared to the bland food condition. Energy intake was also greater in the palatable food condition. In addition, Rogers and Blundell (1990) demonstrated that the consumption of a palatable soup preload before a test meal resulted in a more rapid recovery of hunger compared to when a bland or no preload was consumed. Similarly, research has demonstrated that increased levels of fullness cause a decrease in ratings of pleasantness or liking for foods of a similar sensory

domain (Finlayson, King, & Blundell, 2008; Griffioen-Roose, Finlayson, Mars, Blundell, & de Graaf, 2010) in addition to having an impact on measures of food wanting. For example, Epstein, Truesdale, Wojcik, Paluch, and Raynor (2003) demonstrated in normal weight females that wanting for food was greater in the fasted compared to the fed condition. Furthermore, Finlayson et al. (2008) examined liking and wanting for food in normal weight individuals in a fasted and fed state and demonstrated that while liking for high-fat sweet food decreased in a fed state, implicit wanting (or motivation) for it increased relative to savoury or low fat foods. A difference in wanting for food has also been demonstrated between obese and lean individuals. For example, Castellanos et al. (2009) examined the initial orientation of attention towards palatable food cues and found that attentional bias was greater in obese individuals compared to their lean counterparts independent of motivational state.

The consideration of the interaction between homeostatic and hedonic control of appetite enables researchers to elucidate the role of hedonics in the control and loss of control over food intake (Finlayson, King, & Blundell, 2007). Erlanson-Albertsson (2005) summarise how the ingestion of palatable foods can disrupt the 'normal' homeostatic regulation of appetite. When a standard, moderately palatable food is consumed, information on energy content and taste is generated within the brain stem and transmitted to the hypothalamus, which leads to the release of satiety peptides that act to bring the period of eating to a close. However, following the ingestion of a highly palatable food, taste sensing is different and information is transmitted to the reward circuit, which results in an increase in the level of dopamine and opioids. In response to this, via connections with the appetite-controlling neurons in the hypothalamus, the expression of hunger peptides such as orexin and agouti-related peptide are increased whereas sensitivity is lowered to the satiety signalling peptides like leptin, CCK and insulin which facilitates overconsumption. Therefore, the consumption of highly palatable foods maintains

the motivation to eat, which may lead to overconsumption (Erlanson- Albertsson, 2005).

#### **1.4 Susceptibility to weight gain and overconsumption**

While an obesogenic environment encourages overconsumption, weight gain and obesity, it is apparent that not everyone over-consumes and therefore, there is a large degree of individual variability in the susceptibility to weight gain and obesity. Within a population there is a spectrum of vulnerability – with some individuals being more susceptible or resistant to overconsumption and weight gain than others (Ravussin & Gautier, 1999). Furthermore, along this spectrum it is possible to identify distinct phenotypes that may be characterised by a specific cluster of characteristics (phenotype) or an underlying genotype (Blundell et al., 2005).

Phenotypes susceptible to weight gain and overconsumption may be identified on many different levels, with risk factors encompassing genetic, physiological, metabolic, behavioural and psychological factors (Blundell et al., 2005). Obesity as a result of a single gene mutation is relatively rare, with the most common, a single gene mutation in the MC4R gene, accounting for approximately 4% of adult obesity (Farooqi et al., 2003). However, it is widely agreed that obesity is a condition under polygenic influence (Hinney, Vogel, & Hebebrand, 2010), with genetic susceptibility to weight gain varying greatly among individuals with regards to both the number of obesity related risk alleles and the profile of allelic variation across a number of genes. Several physiological and metabolic factors may enhance vulnerability for weight gain and obesity, including a low basal metabolic rate, low energy cost of physical activity, high insulin sensitivity and a low fat oxidation (Blundell & Finlayson, 2004; Blundell et al., 2005). Finally, a third level of susceptibility relates to behavioural and psychological characteristics. Behaviourally, certain patterns of eating behaviour may increase susceptibility to weight gain, these include, 1) consumption of large meals, 2) frequent eating or grazing behaviours, and 3) enhanced preference for, and consumption of high-fat or energy-dense foods

(Blundell et al., 2005). Further to this, psychological characteristics such as enhanced liking and wanting for food, greater experience of food cravings, and certain eating behaviour traits may also increase susceptibility to over-consumption and obesity. Together, these factors form the bio-psychological approach to investigating susceptibility to overeating and appetite control (see Figure 1.1). This approach takes into consideration the contribution and interaction of different risk factors that may underlie and contribute to increased susceptibility to overconsumption, weight gain and obesity.

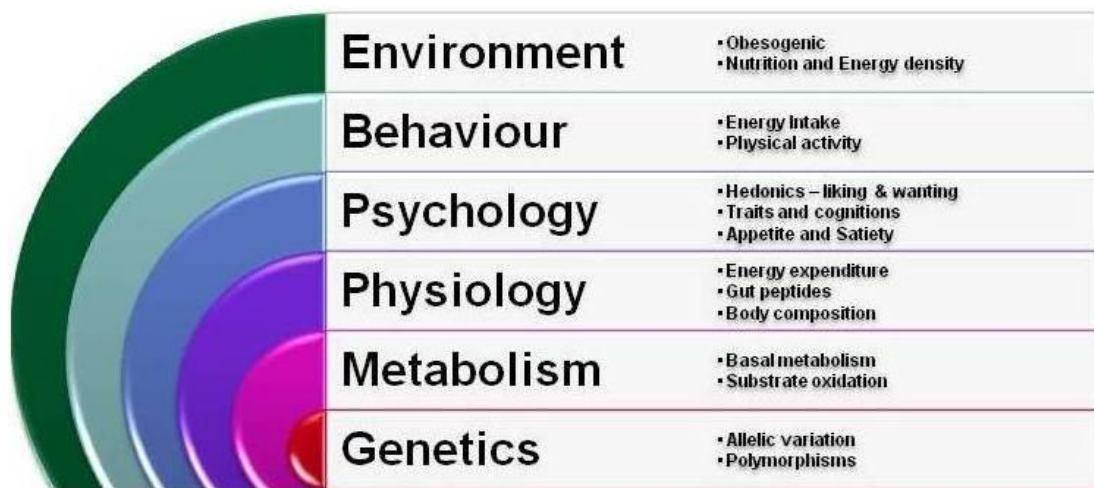


Figure 1.1 Bio-psychological approach to appetite control and energy balance

Using this approach, a series of studies by Blundell and colleagues have distinguished between a high-fat phenotype and low-fat phenotype identified by their habitual fat consumption (Cooling & Blundell, 1998a, 1998b; Macdiarmid, Cade, & Blundell, 1996). Characterisation of these phenotypes revealed that high-fat phenotypes reported higher baseline levels, and quicker recovery of hunger following a meal compared to low-fat phenotypes. Further to this, when provided with ad libitum access to high fat, or high carbohydrate foods, high-fat phenotypes consumed a greater amount of energy from the high-fat foods, compared to low-fat phenotypes, who consumed a similar amount of energy from both types of food (Cooling & Blundell, 1998a). Interestingly, while a greater number of high-fat

phenotypes were overweight or obese, there was a large degree of variability in the distribution of BMI in this phenotype (Macdiarmid et al., 1996) which suggested that although a high habitual fat intake was associated with obesity, some individuals defined as high-fat consumers appeared to be resistant to weight gain. When the mechanisms behind this resistance were investigated, it was shown that the susceptible high-fat phenotypes were characterised by several factors that may be associated with increased susceptibility to weight gain. To begin, the susceptible phenotype showed a weaker suppression of hunger following the consumption of high-fat foods. This effect was not observed following the consumption of low-fat foods, or in the resistant phenotype. Secondly, the susceptible phenotype retained a strong hedonic response to high-fat foods following satiation compared to the resistant phenotype, which exhibited a preference for low-fat foods. Thirdly, the susceptible phenotype scored higher on the trait disinhibition and hunger, which suggested that they might have been more prone to opportunistic eating compared to the resistant phenotype. Finally, the susceptible phenotype reported eating more in response to negative affect whereas the resistant phenotype reported eating less (Blundell et al., 2005).

By identifying, and then characterising distinct phenotypes of obesity it is possible to go beyond the traditional, sweeping classification of obesity in adults as having a BMI equal to or greater than  $30\text{kg/m}^2$ . Obesity is a heterogeneous condition with many distinct phenotypes, characterised by numerous risk factors, behaviours and comorbidities that are not reflected in the traditional BMI definition. This suggests that prevention and treatment strategies may be more effective with a greater understanding of what characterises specific phenotypes.

## Chapter 2<sup>1</sup>

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### **Individual differences in the susceptibility to hedonically driven overconsumption**

#### **2.1 Introduction**

In recent years, there has been a tendency for the hedonic system of appetite control to be considered as having a more predominant role in eating behaviour compared to homeostatic mechanisms. Indeed, reward-driven eating appears to be able to override the inhibitory effects of satiety and drive energy intake beyond energy needs (Berthoud & Morrison, 2008; Blundell & Finlayson, 2004; Erlanson-Albertsson, 2005) and in turn increase susceptibility to weight gain (Blundell & Cooling, 2000). Susceptibility to reward-driven overeating may be characterised by instances where the processes of food reward become enhanced, attenuated or even dissociated to contribute to certain forms of overeating and eating pathology (Finlayson & Dalton, 2012b). Although it cannot be assumed that all instances of overeating are characterised by dysregulated food reward, examining reward based risk factors may help to characterise distinct subtypes within both the normal weight and the overweight or obese population that are vulnerable to reward-driven overeating and weight gain. There were several aims of this chapter; to discuss the evidence from neuroimaging research on the role of food reward in the development of obesity; to provide an overview of current thinking on the neurobiology of food reward and specifically the distinction between liking and wanting as separate psychological components of reward; to introduce techniques of measuring liking and wanting in humans; to examine the role of liking and wanting in eating disorders; and to review the evidence which suggests that liking and wanting may be relevant, and may characterise certain forms of non-clinical disordered eating.

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<sup>1</sup> Parts of this chapter are based on a review article that has been published “Dalton, M., King, N.A., & Finlayson, G., (2013) Appetite, Satiety and Food Reward in Obese Subjects: A Phenotypic Approach, *Current Nutrition Reports*, 1-9.”

## **2.2 Hypo- versus hyper-functioning reward in obesity**

Seemingly incompatible theories have emerged from the neuroimaging literature with regards to the role of reward as a risk factor for overeating, weight gain and the development of overweight and obesity. The first proposes that obese individuals experience a greater amount of reward from food intake as a result of a hyper-functioning reward system (Davis et al., 2007; Davis, Strachan, & Berkson, 2004; Dawe & Loxton, 2004) while the second proposes that obese individuals have a hypo-functioning reward system which causes them to overeat palatable, rewarding foods as a means of compensating for this deficit (Wang et al., 2001; Wang, Volkow, Thanos, & Fowler, 2004).

### **2.2.1 Reward deficit model**

Consistent with the reward deficit model, a landmark positron emission topography (PET) study demonstrated that a small group of extremely obese individuals had reduced striatal dopamine D2 receptor binding compared to lean individuals, with the lowest binding observed in those with the highest BMI (Wang et al., 2001). However, more recently, research regarding dopamine availability and obesity has been less consistent with some research supporting the initial finding (Volkow et al., 2008) and some not (Haltia et al., 2007). However, it has been suggested that differences in the severity of obesity in the samples studied may, in part, account for the discrepancies in findings (Ziauddeen, Farooqi, & Fletcher, 2012b). Two fMRI studies have demonstrated, in line with the reward deficit model, that compared to their lean counterparts, obese adolescents show less activation in the dorsal striatum in response to the consumption of a palatable milkshake versus a tasteless control solution (Stice, Spoor, Bohon, & Small, 2008; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008) however, more recently this finding was not replicated (Ng, Stice, Yokum, & Bohon, 2011). Felsted, Ren, Chouinard-Decorte, and Small (2010) reported that the reduced striatal response to the consumption of a palatable food may be moderated by the Taq1A polymorphism of the dopamine D2 receptor associated ANKK1 gene, as they demonstrated decreased activation in response to

the consumption of chocolate milkshake was only evident in individuals with at least one copy of the A1 allele. The A1 allele of Taq1A polymorphism has previously been associated with a 30-40% reduction in the number of dopamine D2 receptors in the striatum, and weaker dopamine signalling (Noble, Blum, Ritchie, Montgomery, & Sheridan, 1991; Ritchie & Noble, 2003; Thompson et al., 1997).

### **2.2.2 Reward surfeit model**

The reward surfeit model posits that individuals who experience a greater amount of reward from food intake are at risk of overeating (Davis et al., 2007; Davis et al., 2004). Consistent with this model, research has shown that obese individuals demonstrate increased activation of brain regions associated with reward, including the amygdala, striatum, insula and orbitofrontal cortex, in response to images of palatable foods compared to lean controls (Nummenmaa et al., 2012; Rothmund et al., 2007; Stice, Yokum, Bohon, Marti, & Smolen, 2010; Stoeckel et al., 2008). Further to this, the findings from Felsted et al. (2010) suggest that the increased activation to food images may be moderated by the Taq1A A2 allele, which has been associated with increased dopamine availability (Noble et al., 1991; Ritchie & Noble, 2003), as they demonstrated that individuals with the A2/A2 genotype had greater activation in the midbrain and orbitofrontal cortex (OFC) in response to the receipt of milkshake. In addition, Yokum, Ng, and Stice (2011) demonstrated that greater activation in the OFC during initial orientation of attention to appetising food images was associated with an increase in BMI at 1-year follow up in a sample of young females. Behavioural evidence also suggests that obesity is associated with a hyper-responsiveness to reward related cues. For example, Castellanos et al. (2009) found that while both obese and lean individuals exhibited enhanced attentional bias for food cues when fasted, only obese individuals maintained this increased bias for food cues when in a fed state.



### **2.2.3 Dynamic Vulnerability Model**

In an attempt to resolve the opposing models some authors have suggested that a hypo-functioning reward system may be a consequence rather than a cause of obesity in which the dopamine receptors have been down regulated in response to excessive activation (Berridge, Ho, Richard, & DiFeliceantonio, 2010; Davis et al., 2007; Stice & Burger, 2012). Evidence from behavioural neuroscience supports the hypothesis that the repeated ingestion of energy dense foods results in the down-regulation of the dopamine D2 receptors, and is associated with decreased D2 receptor density (Johnson & Kenny, 2010). In humans, Stice, Yokum, Blum, and Bohon (2010) have shown that females who gained weight over a period of 6 months had a marked decline in striatal response to the consumption of palatable food compared to baseline, and compared to females who had remained weight stable. Further to this, recent evidence has shown that adolescents who reported frequent consumption of ice-cream exhibited attenuated reward-region activation specifically in response to the consumption of that food (Burger & Stice, 2012).

The Dynamic Vulnerability Model of obesity posits that individuals at risk for weight gain are initially hyper-responsive to the rewarding aspects of food, which drives overconsumption (Stice & Burger, 2012). The increases in overconsumption and weight gain are proposed to result in a reduction in striatal dopamine activation during food intake. Concurrent with the emergence of a hyposensitive state in response to the consumption of palatable foods, it is proposed that the regions which encode the motivational value of food cues become hyper-responsive leading to increased activation in the anticipation of, but not the consumption of food (Stice & Burger, 2012; Stice, Yokum, Burger, Epstein, & Small, 2011). Therefore, the model suggests that during the development of obesity, the hedonic value obtained from consuming palatable foods would decrease whereas the motivational value of palatable foods (and their associated cues) would increase (Kenny, 2011; Robinson & Berridge, 1993). This hypothesis is supported by studies from behavioural neuroscience (Avena, Long, & Hoebel, 2005; Schultz, Apicella, & Ljungberg, 1993).

In humans, evidence for the model is still preliminary. Yokum et al. (2011) showed that greater responsivity to palatable food cues was associated with a greater amount of weight gain over a period of one year. In a partial test of the model, Stice et al. (2011), demonstrated that adolescents categorised as being at high-risk for the development of obesity (defined as having two parents who were obese) showed greater activation in the somatosensory region in response to the consumption of palatable food compared to adolescents categorised as low-risk, while there were no differences in activation in response to a cue that predicted food intake. From these findings, Stice et al. (2011) have predicted that hyper-responsivity to food cues would develop over time if the individuals over-consumed and gained weight, consistent with the findings of Yokum et al. (2011).

### **2.3 The neurobiology of reward**

For almost two decades, Berridge's (1996, 2007) influential theory of reward has provided a useful framework for investigating the role of hedonics in human appetitive behaviour. The theory posits that reward is not a unitary process but consists of both an affective pleasure component and non-affective motivational component, termed liking and wanting, respectively. The liking component refers to the subjective experience of pleasure obtained from food and is associated with the release of endogenous opioids from localised clusters of neurons termed 'hedonic hotspots' (Peciña, Smith, & Berridge, 2006). To date, hedonic hotspots have been identified in two subcortical regions; the first within the rostradorsal quadrant of the medial shell of the nucleus accumbens and the second in the posterior half of the ventral pallidum (Berridge & Kringelbach, 2013).

Wanting is the motivational component of reward in which incentive salience (or motivational significance) is attributed to rewards and their predictive cues in the environment. The attribution of incentive salience makes the cue and its reward more attractive and desirable, and therefore more likely to be approached (Berridge, 2007). Wanting for food arises through the release of the neurotransmitter dopamine in the

mesocorticolimbic pathway prior to and during contact with food (Berridge, 2007). Berridge (2012) proposed that the motivational value of a cue can vary depending on a number of factors, including, with regards to food reward specifically, level of hunger, time of day and the degree of attentional resources available. To this end, the level of motivation or wanting for a cue is created new on each encounter with it (Berridge, 2012). Therefore, rather than being a constant drive, wanting implies a target with a direction, with the target triggering a cue specific response. Liking and wanting are linked by a third component termed ‘reward learning’, which is important in the initial attribution of incentive value and the linking of liking and wanting responses over time in relation to foods consumed in the diet.

## **2.4 Liking and wanting as psychological components of reward**

The terms ‘liking’ and ‘wanting’ are not only used to refer to the core processes of reward described above but also are discussed in relation to subjective states and objective behaviours that correspond to the more everyday understanding of these terms. Like the core processes of reward identified in behavioural neuroscience, liking and wanting as psychological processes are logically thought to be distinct. However, it is important to make a further distinction between the core processes of liking and wanting, and liking and wanting as psychological constructs, as one cannot infer that the latter is an interpretative read-out of the former. The link between the subjective and behavioural forms of liking and wanting, and the objective neuro-chemical underpinnings are not well understood and providing psychological accounts of pleasure and motivation is a far more complex process that will involve the recruitment of additional brain areas that are related to cognitive evaluations and conscious experience (Berridge & Kringelbach, 2013; Finlayson & Dalton, 2012b).

Importantly, the conceptualisation of liking and wanting as psychological constructs in the current thesis differs from Berridge’s original conceptualisation and is based on the work of Finlayson and colleagues (e.g. Finlayson, Arlotti, Dalton, King &

Blundell, 2011; Finlayson & Dalton, 2012b). Liking is typically understood as the perceived or expected hedonic value of a food, the appreciation of its sensory properties or a judgement of the degree of pleasure it elicits. In this context, liking for food appears to be a relatively enduring trait in an individual, that varies only slightly under specific circumstances. For example, research has shown that liking for food is greater when individuals are in a fasted compared to a fed state (Finlayson, King & Blundell, 2008) and liking for a just eaten food has been shown to decrease in a manner consistent with sensory specific satiation (Griffioen-Roose, Finlayson, Mars, Blundell & de Graaf, 2010). To this end, liking is thought to be more important in determining the range of foods eaten (de Castro, Bellisle, & Dalix, 2000) and in establishing the motivational value of food (Finlayson, King, & Blundell, 2008; Lowe & Levine, 2005).

In contrast, wanting refers to states of desire and craving that are triggered by the food itself or its related cues in the environment. Importantly, rather than being a constant drive, like hunger, the wanting component of reward implies a target with a direction that may vary depending on a number of factors, including level of hunger, time of day and the degree of attentional resources available. Therefore, the level of wanting for food is created new on each encounter with the food or its associated cues. Furthermore, research suggests that the target of wanting can vary from being relatively broad to becoming more focussed. For example, previous research has consistently demonstrated, independent of BMI, that in a fasted state individuals have increased wanting for food in general (Castellanos et al., 2009; Epstein, Truesdale, Wojcik, Paluch, & Raynor, 2003; Hoefling & Strack, 2008; Nijs, Muris, Euser, & Franken, 2010). Further to this, there is some evidence that suggests wanting may become focussed (and at times dissociated from liking) under certain conditions in which one food is wanted to a greater extent than alternatives, such as when individuals are in a state of macronutrient imbalance (Griffioen-Roose et al.,

2012) or in those who exhibit certain eating trait pathologies (Finlayson, Arlotti, Dalton, King, & Blundell, 2011).

The subjective sensations of liking and wanting often overlap and are therefore subject to interference or misinterpretation. For this reason, their relationship with behaviour is often difficult to discern (see Havermans, 2012a; 2012b) and (Finlayson & Dalton, 2012a). However, not all behaviour is under conscious control and liking and wanting responses to food are thought to have both an explicit and an implicit element. For example, people tend to be very good at estimating how much they like a food, yet they find it more difficult to assess their implicit wanting for food (i.e. why they are automatically drawn to one food over another). Therefore, the psychological components of reward have been proposed to operate at implicit (automatic, unconscious) and explicit (subjective, conscious) levels and may bear some relation to dual process models of motivation (Finlayson et al., 2007; Friese et al., 2008; Wilson et al., 2000). The experience of reward typically involves a combination of liking and wanting; however a number of techniques have been developed to measure these components separately in order to examine under which circumstances they differ by degree, or even become dissociated (Finlayson et al., 2007).

## **2.5 Measurement of food reward in humans**

The instantiation of the components of food reward into measurable, psychological constructs is not without its challenges. For a measure of food reward to be plausible it should incorporate the ability to not only reflect the existence of distinct components of reward, but also prevent confounding of one component with another in order to allow for the detection of possible dissociations.

### **2.5.1 Behavioural techniques to assess liking and wanting**

The explicit components of food reward are often measured using visual analogue scales. Questions such as “How pleasant would it be to taste some of this food now?” or “How much do you like this food?” are commonly used for the

measurement of hedonic value or explicit liking whereas questions such as “How strong is your desire to eat this food?” are commonly used for the assessment of explicit wanting. One of the benefits of visual analogue scales is that they are not complicated for the participant. However, self-report techniques may be open to reporting bias due to the impact of social desirability and other methodological issues such as ‘end avoidance’. Nonetheless, when used carefully they can be quite sensitive to experimental manipulations and have been shown to predict food intake behaviour (Fay & Finlayson, 2011; Griffioen-Roose et al., 2010).

Techniques that have been designed to capture the more implicit motivational aspect of food reward typically fall into one of two categories. The first type measures the individuals’ willingness to expend effort in order to obtain a desired target food (Epstein, Leddy, Temple, & Faith, 2007) and the second type measures the individuals’ reaction time of responses to food cues, in which the speed of the response is interpreted as a measure of the motivational value of the cue (Finlayson et al., 2008; Nijs, Muris, Euser, & Franken, 2010). The following section will introduce three commonly used behavioural techniques for the assessment of food reward. The first technique uses a progressive ratio computer task in order to determine the relative reinforcing value of food; the second technique, the Leeds Food Preference Questionnaire, is a computerised based procedure developed to measure both explicit and implicit components of food reward; and the final technique, the visual probe task, is a measure of attentional bias.

#### **2.5.1.1 The Relative Reinforcing Value of food**

The reinforcing value of food is defined as how hard an individual is willing to work to obtain food and is typically operationalised in terms of how many responses are made on a reinforcement schedule to gain access to food. To assess the relative reinforcing value of food a choice methodology is enforced, in which a preferred

food reinforcer and an alternative reinforcer can be worked for in a concurrent schedule paradigm (Epstein & Leddy, 2006).

The alternative reinforcer typically differs across studies, with previous research using healthy snack foods (Goldfield, Adamo, Rutherford, & Legg, 2008), money (Epstein, Dearing, Temple, & Cavanaugh, 2008) and leisure activities such as reading (Epstein, Leddy, et al., 2007) as an alternative to preferred snack foods. The use of healthy snacks has the benefit of the emulating choices that individuals would normally make in their habitual dietary decisions. However, providing an alternative leisure activity reduces the likelihood of the participant responding for the food reinforcer due to boredom (Epstein, Leddy, et al., 2007). One limitation of the use of money as the alternative reinforcer is it creates an open economy where the reward earned is only of benefit outside of the laboratory and therefore may be exchanged for *any* desired food (that is, not limited to the single snack food reinforcer).

Research has demonstrated that the relative reinforcing value of food is influenced by several factors. For example, Raynor & Epstein, (2003) report that level of deprivation positively influenced the reinforcing value of food with participants working harder to obtain a preferred snack food compared to an alternative reinforcer when they were in a food deprived state. In addition, the reinforcing value of food is influenced by variety. Myers and Epstein (2002) reported that when participants were provided with the opportunity to work for a variety of preferred food reinforcers or one preferred food reinforcer, responding for food decreased more rapidly in the one reinforcer condition. In addition, Epstein, Carr, Lin, and Fletcher (2011) demonstrated that increased responding for a preferred snack food over reading was positively related to body mass index, and energy intake assessed in the laboratory and under free-living conditions. These findings suggest that the relative reinforcing value of food is sensitive to experimental manipulations and individual differences.

### **2.5.1.2 The Leeds Food Preference Questionnaire**

The Leeds Food Preference Questionnaire (LFPQ) assesses explicit liking, explicit wanting and implicit wanting for food using an array of photographic stimuli. For the explicit measures individuals are required to provide subjective ratings of the food stimuli according to “*How pleasant would it be to taste some of this food now?*” and “*How much do you want some of this food now?*” to assess explicit liking and explicit wanting, respectively. The LFPQ also includes a forced choice behavioural measure in which individuals are presented with two food images and are required to respond according to “*Which food do you most want to eat now?*” as quickly and as accurately as possible. The speed with which one stimulus is selected in preference to its alternative is an indirect measure designed to assess implicit wanting (Finlayson et al., 2007, 2008).

Previous research has demonstrated that the LFPQ is sensitive to experimental manipulations of motivational state (Finlayson et al., 2008), sensory specific satiation (Griffioen-Roose et al., 2010), and macronutrient imbalance (Griffioen-Roose et al., 2012). Finlayson et al. (2008) reported that moving participants from a fasted to a fed state resulted in a decrease in explicit ratings of liking and wanting for a range of food stimuli varying in taste (sweet or savoury) and fat content (high or low). However, they found that participants’ implicit wanting increased for sweet foods but not for savoury foods in a fed state, suggesting that implicit wanting may be partly dissociable from explicit rating measures. Griffioen-Roose et al. (2012) assessed the impact of a 14-day dietary intervention, in which participants were randomised to consume either a high-protein or a low-protein diet, on ad libitum energy intake (assessed over 2.5 days) and food reward. With regards to food intake, they found that following the low-protein diet participants consumed a significantly greater amount of protein compared to those that followed the high-protein diet. When the outcomes of the LFPQ were examined it was revealed that following the low-protein diet, explicit liking and wanting were enhanced for savoury foods,



whereas ratings following the high-protein diet remained stable. Further to this, following the low-protein diet implicit wanting was enhanced for high protein foods – this effect was consistent with the participants’ actual eating behaviour but was not observed in the explicit measures of reward. The authors suggested that when an individual is in macronutrient balance, the explicit and implicit responses to food are similar. However, when a state of macronutrient imbalance is introduced, implicit processes appear to exact a stronger determining role on what is eaten (Griffioen-Roose et al., 2012).

### **2.5.1.3 Attentional bias for food**

Attentional bias is defined as the tendency to favourably attend to salient information in the environment over less salient or more neutral information (Mathews & MacLeod, 2005). The visual probe task was developed by MacLeod, Mathews, & Tata, (1986) as a measure of attention bias in emotional disorders. In the task, participants are briefly presented with an image pair comprising one salient image and one matched control. A probe, which is either an upward or downwards facing arrow or a single dot depending on the type of visual probe task being used, then replaces the image pair. Participants are required to identify the location of the dot or to specify whether the arrow is pointing upwards or downwards. Reaction times are faster when the probe replaces the attended to image. An attentional bias for salient information is apparent when reaction times are faster when the probe replaces the salient image compared to the control image. Using the visual probe task, attentional bias can be assessed for both the initial orientation of attention and the maintenance of attention depending on the exposure times of the image pair.

Attentional bias for food stimuli is determined by the hedonic value (or motivational salience) of the food stimulus compared to a control image and previous research has demonstrated that this value can be modulated by internal motivational state and BMI (Castellanos et al., 2009; Nijs et al., 2010). For example, Nijs et al. (2010) examined attentional bias for high-calorie food images in a sample of overweight or

obese and normal weight females. They found that while all participants exhibited an enhanced attentional bias for food when in a fasted compared to a fed state, this effect was stronger in those who were overweight or obese. Furthermore, Yokum et al. (2011) examined attentional bias for appetising compared to unappetising or non-food images in conjunction with functional magnetic resonance imaging (fMRI) in young females ranging from lean to obese. They found that BMI was positively associated with enhanced orientation of attention towards food compared to non-food images. In addition, longitudinal analyses of change in BMI after 1-year revealed that activation in the orbitofrontal cortex during initial orientation to appetising food images was correlated with an increase BMI indicating that increased responsiveness to food cues results in an increased risk for future weight gain.

### **2.5.2 Functional neuroimaging techniques**

Functional neuroimaging techniques allow for the study of patterns of brain activation that are associated with cognitive and behavioural processes. The term encompasses a number of brain imaging methodologies, including functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). One commonly used procedure in neuroimaging research is to examine regional differences in neural activation to the consumption (consummatory reward) or the anticipated consumption (anticipatory reward) of a rewarding stimuli compared to neutral stimuli (usually chocolate milkshake versus a tasteless solution, respectively) (Stice, Spoor, et al., 2008a; Stice, Spoor, Ng, & Zald, 2009). Regional differences in neural activation to food stimuli during the anticipatory or the consummatory phase of reward processing are broadly supportive of the distinction between wanting and liking for food, respectively. For example, increases in neural activation in response to exposure to high compared to low calorie food images or to a cue that signals the receipt of a palatable food or drink versus a tasteless solution have been more consistently observed in the amygdala and ventral striatum (Beaver et al., 2006; Small, 2009; Small, Veldhuizen, Felsted, Mak, & McGlone, 2008). Conversely, an

increase in neural activation in response to the consumption of a palatable food has been more consistently observed in the orbitofrontal cortex and the insula (Kringelbach, de Araujo, & Rolls, 2004; O'Doherty, Deichmann, Critchley, & Dolan, 2002). However, it is important to note that a large degree of inconsistency exists in the fMRI literature with regards to obesity and food reward (Ziauddeen et al., 2012b).

In addition to differences in activation in response to the signal or receipt of a palatable food, evidence from neuroimaging research has also demonstrated the interaction between the homeostatic and the hedonic systems of appetite control. For example, Goldstone et al. (2009) compared activation to high versus low calorie food images in normal weight adults who were either in a fasted or fed state. They found that in the fasted state, high calorie food images selectively increased activation in the amygdala, anterior insula and lateral OFC. In addition, Goldstone et al. (2009) demonstrated that subjective ratings of food liking were increased for high calorie versus low calorie food images in the fasted compared to the fed state. Furthermore, and consistent with the research outlined above, this greater liking for high calorie food images was positively associated with activation in the OFC.

## **2.6 The role of food reward in eating disorders**

### **2.6.1 Anorexia nervosa**

Anorexia nervosa (AN) is characterised by restricted eating, distorted body image and an intense fear of weight gain (APA, 2000). Contrary to healthy individuals, exposure to, and consumption of food provokes feelings of anxiety and dysphoric mood in individuals with AN (Kaye et al., 2013; Steinglass et al., 2010). This is in line with evidence from a recent study that compared responses on a behavioural measure of liking and wanting for high and low calorie foods across three groups of individuals with AN (current AN, weight restored AN and recovered AN) and healthy controls (Cowdrey, Finlayson, & Park, 2013). They demonstrated that compared to healthy controls, current AN and weight restored AN had lower explicit

liking and implicit wanting for high-calorie foods whereas implicit wanting for low-calorie foods was greater in these individuals. These findings suggest that AN is characterised by a decreased motivation for potentially anxiety-inducing high-calorie foods. In a recent meta-analysis of the literature on self-report measures of food reward, Harrison, O'Brien, Lopez, and Treasure (2010) found that individuals with AN scored higher in sensitivity to punishment and low in novelty seeking compared to healthy controls. In addition, they found that differences emerged with regards to reward responsiveness, with the binge-purge subtype exhibiting higher reward responsiveness scores and the restricting subtype exhibiting lower reward responsiveness scores. Further to this, Farmer, Nash, and Field (2001) demonstrated that reward sensitivity was positively related to purging frequency.

Evidence from neuroimaging studies suggests that individuals with AN are highly responsive to food related cues in the ventral striatum but also display increased activation in areas associated with impulse control. For example, in an fMRI study, Cowdrey, Park, Harmer, and McCabe (2011) compared the neural response to the sight and taste of a pleasant and an aversive stimulus in a sample of recovered AN individuals and healthy controls. They found there were no differences in subjective ratings for the stimuli between groups however the recovered AN exhibited an increase in neural response to both rewarding and aversive food stimuli. Further to this, Frank et al. (2012) demonstrated that compared to healthy controls, individuals with AN displayed enhanced activation in the lateral orbitofrontal cortex in response to the receipt of an unexpected food reward. Taken together these findings suggest that the increased responsiveness individuals with AN display to food-related cues may reflect a mechanism by which they are able to predict and control the onset of anxiety that is caused by food. This suggestion is supported by the enhanced activation in the lateral orbitofrontal cortex, which may help individuals with AN to maintain high levels of dietary restraint (Kaye et al., 2013).

### **2.6.2 Bulimia nervosa**

Bulimia nervosa (BN) is characterised by recurrent episodes of binge eating large quantities of food followed by inappropriate compensatory measures such as purging in order to prevent weight gain (APA, 2000). Previous research suggests that individuals with BN are more impulsive and have impaired self-regulatory control compared to individuals with AN and healthy controls (Rosval et al., 2006; Uher et al., 2004; Z. Wang et al., 2009). For example, Rosval et al. (2006) demonstrated that individuals with BN displayed impaired response inhibition on a Go/No-Go computer task and scored higher on the Barratt Impulsivity scale. Further to this, studies examining attentional bias using the emotional Stroop task have shown that individuals with BN exhibit increased attentional interference to food or weight related words compared to healthy controls (Davidson & Wright, 2002; Lokken, Marx, & Ferraro, 2006). While research using the Stroop task cannot ascertain whether attentional interference arises from selective attention towards the stimulus or from attentional avoidance of it further research suggests that individuals with BN use strategies to avoid processing salient food stimuli. For instance, in an fMRI study, Brooks et al. (2011) compared the neural response to food versus non-food images in a sample of individuals with BN and healthy controls and found that individuals with BN had a reduced visual cortex response to food images. In line with this, Schienle, Schafer, Hermann, and Vaitl (2009) demonstrated that when exposed to palatable food images individuals with BN displayed greater subjective ratings of arousal and activation in the anterior cingulate cortex and the insula. The authors suggest that increased activation in these areas may reflect their attempts to counter-regulate the increased arousal and desire they report for food.

### **2.6.3 Binge eating disorder**

Binge eating disorder (BED) was initially introduced in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) as a provisional eating disorder diagnosis that required further study. Despite some concerns (Stetka & Correll, 2013), the recent release of the fifth edition of the DSM sees BED being

reassigned as an official eating disorder. BED is defined by recurrent episodes of binge eating in the absence of inappropriate compensatory behaviours, coupled with feelings of loss of control and significant distress over eating. Previous research has demonstrated that compared to their weight matched counterparts, individuals with BED consume significantly more food in ad libitum test meals (Geliebter, Hassid, & Hashim, 2001; Goldfein, Walsh, Devlin, Lachaussee, & Kissileff, 1993; Latner, Rosewall, & Chisholm, 2009; Yanovski et al., 1992) and consume more energy from fat (Yanovski & Sebring, 1994) and carbohydrates (Wallin, Norring, & Holmgren, 1994).

BED has been proposed to be a subtype of obesity that is characterised by hyper-responsiveness to the hedonic properties of food (Davis, Levitan, Kaplan, et al., 2008; Davis et al., 2009; Davis et al., 2012; Nasser, Evans, Geliebter, Pi-Sunyer, & Foltin, 2008). For example, Nasser et al. (2008) demonstrated that obese individuals with BED had greater food reinforcement in a fed state compared to obese individuals without BED. Further to this, Svaldi, Tuschen-Caffier, Peyk, and Blechert (2010) used electroencephalography (EEG) to examine attentional bias for high and low calorie food images in overweight or obese females with and without BED. They found that individuals with BED had increased electrophysiological activity in response to high calorie food images compared to individuals without BED. In an fMRI study, Schienle et al. (2009) demonstrated that individuals with BED had greater trait sensitivity to reward scores compared to individuals without BED. Furthermore, individuals with BED had enhanced activation in the medial orbitofrontal cortex, an area that has previously been associated with drug craving and processing of reward value (Kringelbach & Rolls, 2004; Wang et al., 2004).

The notion that BED is a distinct subtype of obesity characterised by a hyper-functioning reward system is further supported by studies that examine genetic variation in the opioid mu-receptor (OPRM1) gene and genes that encode for, or are associated with the dopamine D2 receptor (DRD2). Davis et al. (2009) examined

psychological and genetic markers of hedonic eating in individuals with and without BED. Participants were genotyped for the rs1799971 and the rs1800497 polymorphisms of the OPRM1 gene and the DRD2 related Taq1A gene, respectively. It was demonstrated that a greater proportion of individuals with BED had the A2/A2 genotype of rs1800497 and at least one copy of the rs1799971 G allele – both variants are associated with greater functionality. This finding was replicated in a study by Davis et al. (2012) who demonstrated that a greater number of individuals with BED were homozygous for the A2 allele of rs1800497. Furthermore, they found a greater frequency of the T/T genotype of the DRD2 gene polymorphism rs6277 in the BED group compared to obese controls. In addition, individuals with the A2/A2 genotype reported experiencing greater food cravings compared to those with at least one copy of the A1 allele (Davis et al., 2012).

#### **2.6.3.1 ‘Food addiction’?**

‘Food addiction’ has recently been proposed as a valid subtype of obesity, with some researchers suggesting that individuals with BED may be addicted to food (Davis & Carter, 2009; Davis, Curtis, et al., 2011; Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt et al., 2011). While the debate over whether BED should be redefined as a form of addiction has been present in the scientific literature for over two decades (Cassin & von Ranson, 2007; Davis & Carter, 2009; Davis & Claridge, 1998; Davis, et al., 2011; Gearhardt, et al., 2011; Haddock & Dill, 2000; Wilson, 1991, 2010) it is only more recently that the notion of ‘food addiction’ as a valid and genuine biopsychological disorder has gained momentum as a controversial social, political and scientific issue. This is perhaps due in part to advances in the behavioural neuroscience field in which evidence suggests that rats maintained on a high sugar diet display behaviours that are consistent with several behavioural indicators of substance dependence when the diet is withdrawn (Avena & Hoebel, 2003; Avena, Long, & Hoebel, 2005; Avena, Rada, & Hoebel, 2008, 2009; Hoebel, Avena, Bocarsly, & Rada, 2009; Hoebel, Rada, Mark, & Pothos, 1999). Further to this, the

development of the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin, & Brownell, 2009), based on the DSM-IV criteria for substance dependence, has provided the means for the investigation of dependence-like behaviours in humans.

Research suggests that the YFAS distinguishes a more severe subtype of BED, one that is associated with greater levels of negative affect, lower self-esteem and more frequent binge eating episodes (Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt, et al., 2011). A study by Davis, et al., (2011) demonstrated that obese individuals who met the diagnostic criteria for food addiction had greater co-morbidity with BED. In addition they found that obese individuals with BED *and* food addiction were more impulsive, experienced greater depressive symptoms and reported greater food cravings than those with BED without food addiction. More recently, Davis et al. (2013) utilised a novel genetic methodology in which five functional genetic variants that have previously been associated with the dopaminergic system were combined to create a multi-locus genetic profile score (MLGP) in order to examine whether variants associated with greater dopamine signalling distinguished between individuals with and without food addiction. They found that participants with food addiction had a greater MLGP score indicating they had enhanced dopamine signalling. Furthermore, the association between food addiction and the MLGP score was mediated by eating behaviours associated with elevated responsiveness to palatable foods, including binge eating.

The validity of the concept of food addiction in humans has been questioned by a number of authors (Blundell & Finlayson, 2011; Ziauddeen et al., 2012b; Ziauddeen & Fletcher, 2012) and at the present time is a source of debate (Avena, Gearhardt, Gold, Wang, & Potenza, 2012; Ziauddeen, Farooqi, & Fletcher, 2012a). However, one consensus that has emerged recently is that if food addiction is a valid phenomenon is it likely to only apply to an extremely small subset of individuals whose behaviour resembles that of substance dependence – such as in the case of individuals with severe BED (Davis & Carter, 2009; Davis et al., 2013).



## **2.7 The role of food reward in eating behaviour traits**

### **2.7.1 Impulsivity**

Impulsivity is a personality trait that has been defined, and can be measured in a number of ways. Therefore, impulsivity is generally thought to be a multidimensional construct. A review by Guerrieri, Nederkoorn, and Jansen (2008) identified several common constructs in the different models of impulsivity, of which two have been frequently related to overeating and obesity (Guerrieri et al., 2007; Nederkoorn, Jansen, Mulken, & Jansen, 2007). The first is a heightened sensitivity to reward, which may be operationalised behaviourally using experimental procedures such as delay discounting (Davis, Patte, Curtis, & Reid, 2010) and the Iowa Gambling task (Guerrieri, Stanczyk, Nederkoorn, & Jansen, 2012) or psychometrically through the use of self-report questionnaires (Carver & White, 1994; Davis et al., 2004). The second is a reduced ability to inhibit an inappropriate response, which can be assessed behaviourally using delay of gratification and go/no-go tasks (Jasinska et al., 2012; Seeyave et al., 2009).

#### **2.7.1.1 Sensitivity to reward**

Sensitivity to reward has been defined as a psychobiological trait that is associated with increased vulnerability for disordered eating behaviours (Davis et al., 2007; Franken & Muris, 2005). Franken and Muris (2005) demonstrated that greater sensitivity to reward scores, assessed using the Sensitivity to Punishment and Sensitivity to Reward questionnaire (Torrubia, Avila, Moltó, & Caseras, 2001), were positively associated with BMI and the experience of food cravings in a sample of normal weight females. Davis et al. (2007) also demonstrated that individuals with greater sensitivity to reward scores reported a greater preference for foods high in fat and sugar. Further to this, in an fMRI study, Beaver et al. (2006) reported that individual differences in sensitivity to reward were associated with increased activation to palatable food images (relative to bland food images) in regions previously associated with reward including the ventral striatal, amygdala, midbrain,

orbitofrontal cortex, and ventral pallidum. More recently, Guerrieri et al. (2012) examined the association between reward sensitivity, level of food variety and feelings of hunger on energy intake. They found that individuals high in reward sensitivity who also reported experiencing greater feelings of hunger in a varied food environment consumed more energy compared to individuals in the same condition but who had low reward sensitivity.

#### **2.7.1.2 Impaired response inhibition**

Impulsivity is also characterised by impairments in inhibitory control in which the ability to stop or suppress a response that is inappropriate or in conflict with current goals is diminished (Verbruggen & Logan, 2009). In an fMRI study by Batterink, Yokum, and Stice (2010), the neural activation during a food-based go/no-go task was examined in a sample of adolescent females ranging from lean to obese. Participants were instructed to press a button (“go”) to all images of vegetable items but to withhold their responses to images of dessert items (“no/go”). It was demonstrated that higher BMIs were associated with greater levels of behavioural impulsivity and reduced activation of frontal inhibitory regions including the medial prefrontal and orbitofrontal cortex when instructed to inhibit a predominant response (Batterink et al., 2010). Further to this, Nederkoorn, Houben, Hofmann, Roefs, and Jansen (2010) demonstrated that greater implicit preference for snack foods coupled with low inhibitory control was associated with greater weight gain over 1-year in a sample of undergraduate students which suggests that poor inhibitory control is associated with greater energy intake which may lead to increases in body weight. In line with this suggestion, Houben (2011) experimentally manipulated inhibitory control using a Stop Signal Task. In the control condition, they found that individuals with low levels of inhibitory control consumed more food in a bogus taste test compared to individuals with high levels of inhibitory control. However, when inhibitory control was manipulated to be high, the energy intakes of

participants with previously low levels of inhibitory control were reduced to a similar amount consumed by those with high levels of inhibitory control.

### **2.7.1.3 Interaction between high sensitivity to reward and impaired inhibitory control**

As discussed in the previous sections, sensitivity to reward and inhibitory control are implicated in energy intake and obesity, however susceptibility to weight gain is more than likely influenced by an interaction of both of these constructs (Ely, Winter, & Lowe, 2013). For instance, elevated reward sensitivity may not only increase motivation to eat but may also make it difficult to inhibit the urge to consume palatable foods. In line with this, Appelhans et al. (2011) found that high food reward sensitivity predicted energy intake in the absence of hunger, but only when participants were also low in inhibitory control. Furthermore, a study by Rollins, Dearing, and Epstein (2010) demonstrated, in line with previous research, that individuals who worked harder for a palatable snack food relative to an alternative consumed more energy in an ad libitum taste test. In addition, this effect was moderated by the degree of delay discounting, which is a measure of the devaluation of a reward over time that assesses both instant gratification and response inhibition (Reynolds, 2006). They found that individuals who showed higher discounting of future rewards and increased relative reinforcing value of food consumed more energy than individuals who showed lower discounting and a comparable level of food reward.

### **2.7.2 Dietary restraint**

Individuals defined as restrained eaters are characterised by their intent to restrict their food intake, with a specific emphasis on the restriction of unhealthy or ‘tempting’ foods in order to maintain control over body weight (Herman & Mack, 1975). However, restrained eaters are often unsuccessful in their attempts to restrict their intake and often engage in overeating (Fedoroff, Polivy, & Herman, 1997; Jansen & Van den Hout, 1991). The Goal Conflict Theory (Stroebe, Papies, & Aarts,

2008; Stroebe, van Koningsbruggen, Papies, & Aarts, 2012) proposes that the limited success of restrained eaters attempts to control their intake is the result of them holding two incompatible goals; the goal of controlling their weight and the goal of eating enjoyment. The theory posits that the weight control goal is of paramount importance to the restrained eater as it is associated with greatly desired consequences. While the weight control goal is predominant, it can be inhibited by the eating enjoyment goal, which refers to the anticipation and expectation of the experience of pleasure that consuming a desired palatable food would bring. The eating enjoyment goal is typically activated by exposure to food cues within the environment and once activated can result in overeating. The weight control goal can be reinstated by exposure to diet-relevant cues such as calorie information or changes in the way one's clothes fit. For example, Papies, Stroebe, and Aarts (2008) demonstrated that following activation of their eating enjoyment goal (via pre-exposure to food cues) restrained eaters displayed an enhanced attentional bias for highly palatable food cues compared to unrestrained eaters. However when restrained eaters were primed with diet-congruent cues there was no difference in attentional bias scores for low-palatable or high-palatable foods.

The increased sensitivity to the hedonic aspects of food observed in restrained eaters appears to be in part attributable to an increase in wanting as opposed to an increase in liking for food (Giesen, Havermans, & Jansen, 2010; Giesen, Havermans, Nederkoorn, Strafaci, & Jansen, 2009; Hoefling & Strack, 2008; Veenstra & de Jong, 2010). For instance, a study by Giesen et al. (2009) examined the relative reinforcing value of a high calorie compared to a low calorie snack food in restrained and unrestrained eaters. They found that compared to unrestrained eaters, restrained eaters worked harder to obtain the high calorie snack food. These findings were extended by Veenstra and de Jong (2010) who compared restrained and unrestrained eaters liking and wanting (assessed using the Affective Simon Manikin Task as a measure of automatic approach tendencies) for high fat compared to low-fat foods.

They found that while restrained and unrestrained eaters had a similar liking for high-fat foods, restrained eaters showed greater approach tendencies for both high-fat and low-fat food. The findings that restrained eating is characterised by increases in wanting as opposed to liking is supported by studies that have shown restrained eaters report a greater experience of food cravings compared to unrestrained eaters, especially for restricted foods (Fedoroff, Polivy, & Herman, 2003; Polivy, Coleman, & Herman, 2005). Furthermore, Fedoroff et al. (2003) demonstrated that following exposure to the smell of pizza, or cookies, restrained eaters reported greater food cravings and consumed more energy compared to unrestrained eaters. Importantly, restrained eaters only consumed more energy when the cued food and the presented food were the same. This finding supports the notion that wanting is a specific cue-triggered response rather than being a general desire to eat.

### **2.7.3 Disinhibition**

Disinhibition is commonly assessed using the Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) and reflects the tendency to eat opportunistically and is characterised by increased responsiveness to palatable food cues (Bryant, King, & Blundell, 2008). Greater levels of disinhibition have been consistently associated with increased energy intake (Chambers & Yeomans, 2011; Ouwens, van Strien, & van der Staak, 2003; M.R. Yeomans, Tovey, Tinley, & Haynes, 2004), greater BMI (Blundell et al., 2005; Dykes, Brunner, Martikainen, & Wardle, 2003) and increased propensity for weight gain (Carr, Lin, Fletcher, & Epstein, 2013; Finlayson, Cecil, Higgs, Hill, & Hetherington, 2012; Hays & Roberts, 2008; Wing et al., 2008).

Disinhibition has been demonstrated to moderate the relationship between food reinforcement and energy intake, and food reinforcement and BMI (Epstein, Lin, Carr, & Fletcher, 2012). Epstein et al. (2012) demonstrated that BMI and energy intake were greatest in individuals who were high in both food reinforcement (assessed using the relative reinforcing value of food) and disinhibition. Further to

this, Carr et al. (2013) examined whether the effect of food reinforcement on weight gain over the course of twelve months was moderated by disinhibition in a sample of non-obese adults. They found that while greater levels of food reinforcement predicted weight gain, weight gain was the greatest in individuals who had high levels of food reinforcement *and* greater disinhibition scores.

#### **2.7.4 Binge eating**

Binge eating behaviour is typically characterised by the excessive consumption of food that is not driven by hunger or energy need (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007) and is often accompanied by feelings of guilt and loss of control over eating (Ricca et al., 2009). The tendency to binge eat is observed in both clinical and non-clinical populations, with the distinction between them being the magnitude, frequency and the associated distress of the binge eating episodes (De Zwaan et al., 1994; Hudson, Hiripi, Pope Jr, & Kessler, 2007).

Independent from a clinical diagnosis of BED, recurrent episodes of binge eating are estimated to occur in 10-20% of the general obese population (Spitzer et al., 1993; Striegel-Moore et al., 2009) and constitute a trait that can be assessed psychometrically and applied to the general population. Importantly, the trait of binge eating, assessed by a validated psychometric questionnaire, occurs in normal, overweight, and obese individuals. The psychological tendency to binge eat has been proposed as a psycho-marker for reward driven overeating that may constitute a risk factor for weight gain (Davis, 2009; Davis, Levitan, Carter, et al., 2008; Finlayson et al., 2011; Finlayson & Dalton, 2012b). A study by Finlayson, Cecil, et al. (2012) supports this notion, as they found that trait binge eating was positively associated with increases in fat mass over a period of one year in a sample of first year undergraduate students.

In an fMRI study, Filbey, Myers, and DeWitt (2012) examined the neural activation in response to a cue that signalled the delivery of a preferred high-calorie drink in a sample of overweight or obese individuals with moderate levels of binge eating

severity. They found that exposure to high-calorie taste cues elicited activation in reward related regions, including the amygdala, insula and putamen. Further to this, they found that this activation was moderated by binge eating severity, as activation was greatest in individuals with higher binge eating scores suggesting that these individuals may be hyper-responsive to cues that signal delivery of a preferred high-calorie drink. However, the findings from this study are somewhat limited due to the lack of a non-binge eating control group. A study by Finlayson et al. (2011) used the Binge Eating Scale (BES; Gormally, Black, Daston, and Rardin, 1982) to assess binge-eating severity in a sample of normal-weight females. They demonstrated that higher scores on the BES were associated with enhanced liking ratings for all foods and a specific increase in wanting for high fat sweet foods. Furthermore, the enhanced wanting for sweet foods in the higher scorers coincided with them consuming 50% more high fat sweet foods in an ad libitum test meal. These findings are supported by research that suggests binge-eating behaviour is associated with increased cravings for sweet foods (Greeno, Wing, & Shiffman, 2000; Kampov-Polevoy, Alterman, Khalitov, & Garbutt, 2006) and the experience of loss of control over eating (Ricca et al., 2009). It is plausible therefore that wanting is involved in increasing energy intake via its influence on food choice (Mela, 2006). Finlayson et al. (2011) also demonstrated that higher trait binge eating scores were related to a smaller suppression of hunger by a fixed energy preload. This finding complements previous research that has shown weakened satiety signaling and elevated levels of hunger are characteristic of individuals with binge eating tendencies (Sysko, Devlin, Walsh, Zimmerli, & Kissileff, 2007). In addition Nasser, Geliebter, and Pi-Sunyer (2005) reported that greater BES scores were associated with higher levels of food reinforcement after a preload had reduced hunger.

## **2.8 Summary**

The processes of liking and wanting have their neural correlates in animals (and humans) and progress has been made in distinguishing and measuring the

psychological components of liking and wanting in humans. The research presented in this review suggest that instances where the processes of liking and wanting become either enhanced or dissociated can be observed in eating disorders and in certain forms of non-clinical disordered eating identified by eating behaviour traits – which may have relevance for identifying susceptibility to overeating in the general population. To this end, while the term ‘binge eating’ is often synonymous with BED in the scientific literature, evidence suggests that trait binge eating may be a reliable psychobiological marker for susceptibility to overeating and a phenotype from which to investigate individual differences in underlying processes of reward, appetite and physiology in normal weight and obese adults.



## Chapter 3

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### Aims and objectives

#### 3.1 Aim of thesis

For most people food is a reliable source of pleasure and the hedonic system of appetite control is considered to have a primary role in eating behaviour, able to override the inhibitory effects of satiety signals and drive food intake beyond energy needs. It can be argued that some individuals derive more pleasure from eating than others, and that some are more responsive to food-related stimuli within the environment. Therefore, instances where the hedonic processes of appetite control become either attenuated or enhanced may characterise certain forms of disordered eating behaviour. The aim of this thesis was to investigate the role of hedonic processes (liking and wanting) in individuals identified as being susceptible to overeating.

#### 3.2 Specific objectives

- To examine the association between two reaction time based behavioural measures of food wanting.
- To determine whether trait binge eating identifies a phenotype susceptible to overeating in lean individuals.
- To determine whether trait binge eating identifies a distinct phenotype in overweight or obese individuals.
- To examine the role of liking and wanting for food as risk factors for overeating in trait binge eating and to determine other potential markers of susceptibility in this phenotype.
- To determine whether the behaviours and processes observed under laboratory conditions apply in a natural setting.
- To examine the impact of potential genetic markers for susceptibility to overeating and weight gain on eating behaviour, body composition and food hedonics.

## Chapter 4

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### General Methodology

#### 4.1 Ethical considerations

Ethical approval for each study was obtained from the Board of Ethics at the Institute of Psychological Sciences, University of Leeds. With regards to Chapter 9, ethical approval was also obtained from the Board of Ethics at the School of Medicine, University of St. Andrews. Each study met the ethical requirements of the Institute and followed the code of ethics and conduct of the British Psychological Society (2009). The informed consent of all participants was obtained prior to the commencement of each study. While all study procedures were explained to participants in advance of obtaining their consent, the specific objectives of the studies were not disclosed until participants were debriefed in order to reduce demand characteristics. Participants were informed of their right to withdraw from a study without having to provide a reason. Upon completion of a study, participants were debriefed and given the opportunity to ask questions. In all studies, participants received either course credits or a small monetary payment as compensation for their time.

#### 4.2 Psychometric questionnaires

For the purposes of examining individual differences and psychological traits, each study in the current thesis used a number of validated psychometric questionnaires. The questionnaires selected for each study were tailored to its objectives. Each psychometric questionnaire used in the current thesis is described in detail below.

##### 4.2.1 Binge Eating Scale

Developed by Gormally et al. (1982), the Binge Eating Scale (BES) measures the severity of binge eating and is comprised of sixteen items; eight items describe the behavioural manifestations of binge eating behaviour and eight items describe the feelings and cognitions associated with binge eating. Each item consists of three to

four descriptive statements that increase in severity (e.g. *“I don’t have any difficulty eating slowly in the proper manner”* to *“I have the habit of bolting down my food without really chewing it. When this happens I usually feel uncomfortably stuffed because I’ve eaten too much”*). Participants were required to select the statement from each of the sixteen items that was most descriptive of them. Scores were summed to produce a total score ranging from 0 – 46. Cut off points have previously been reported denoting mild ( $\leq 17$ ), moderate (18-26) and severe ( $\geq 27$ ) binge eating behaviour (Marcus, Wing, & Hopkins, 1988). The BES has been shown to have good internal validity, with a Cronbach’s alpha of 0.89 (Freitas, Lopes, Appolinario, & Coutinho, 2006) and good test-retest reliability (Timmerman, 1999). In the current thesis, Cronbach’s alpha for the entire scale was .88. Since its development the BES has been used in a wide range of research to measure binge-eating severity. In the current thesis, the BES was used in Chapters 6 through to Chapter 9 and was used in order to assign participants to high or low binge eating groups (Chapters 6, 7 and 9) or to recruit participants on the basis of their binge eating score (Chapter 8).

#### **4.2.2 Three Factor Eating Questionnaire**

The Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) is a 51-item scale that assesses three aspects of eating behaviour; cognitive control of restraint (21 items;  $\alpha = .80$ ), disinhibition of eating (16 items;  $\alpha = .75$ ) and susceptibility to hunger (14 items;  $\alpha = .66$ ). Participants were required to respond either true or false to the first 36-items, whereas the remaining items required participants to select a response from a choice of four that varied in the level of agreement with a particular statement. Responses were scored 0 or 1 and summed, with higher scores denoting higher levels of eating disturbances. The TFEQ has been shown to have good internal validity (Stunkard & Messick, 1985) and in the current thesis, Cronbach’s alpha for the entire scale was .78. The subscales of TFEQ were used as dependent variables in Chapters 6 through to Chapter 8, and as covariates, where applicable, in Chapter 9.

#### **4.2.3 Behavioural Activation Scale**

The Behavioural Activation Scale (BAS) of the Behavioural Inhibition Scale/Behavioural Activation Scale (Carver & White, 1994) was used to measure sensitivity to reward. The BAS assesses three personality measures related to reward sensitivity; BAS fun seeking (4 items;  $\alpha = .72$ ), BAS drive (4 items;  $\alpha = .80$ ) and BAS reward responsiveness (5 items;  $\alpha = .74$ ). The BAS fun seeking subscale measures the degree to which an individual has the desire to experience new rewards, and their willingness to approach these. The BAS drive subscale consists of statements assessing the tendency to pursue desired goals. Finally, the BAS reward responsiveness subscale focuses on the experience of positive responses to the occurrence and anticipation of reward. Research has suggested that the three BAS subscales do not form a unitary measure of appetitive motivation and should therefore be treated as separate subscales (Ross, Millis, Bonebright, & Bailey, 2002). The BAS drive and BAS reward responsiveness subscales have been shown to have better internal validity and reliability than the BAS fun seeking subscale (Carver & White, 1994). The subscales of the BAS were used as dependent variables in Chapter 9.

#### **4.2.4 Yale Food Addiction Scale**

The Yale Food Addiction Scale (YFAS; Gearhardt, Corbin, & Brownell, 2009) is a single factor questionnaire composed of 25-items designed to measure the tendency to engage in addictive eating behaviours with high fat and/or sugar foods. The questions are designed to form the seven criteria of symptoms for diagnosis of substance abuse as specified in the Diagnostic and Statistical Manual of Mental Disorders IV-R (e.g. *“I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developed”* to assess symptoms of withdrawal). Participants were asked to respond with regards to the occurrence of addictive-like eating behaviours during the previous 12 months. The YFAS can be scored in one of two ways; firstly, a “symptom” count indicates the number of addictive eating behaviours experienced in the past 12 months (ranging from 0 – 7),

and secondly, a “diagnostic” threshold that requires three or more symptoms to be present in addition to the participant reporting significant impairment or distress as a result of their eating behaviour (Gearhardt et al., 2011). The YFAS has been shown to have adequate internal reliability, and incremental validity in predicting binge eating (Gearhardt et al., 2009). In the current thesis, the YFAS was used in Chapter 8 for the purposes of examining whether individuals scoring high in trait binge eating could be further subtyped into two groups with differing degrees of eating pathology.

#### **4.2.5 Control of Eating Questionnaire**

The Control of Eating Questionnaire (COEQ; Hill, Weaver & Blundell, 1991) comprises 21 items divided into 6 sections. Participants were required to answer according to their experience over the previous seven days. The first two sections measure general appetite and mood. The third and fourth sections assess the frequency and intensity of food cravings in general – with food cravings being defined as a “strong urge to eat a particular food or drink”. The fifth section concerns cravings for specific foods (e.g. dairy, sweet or savoury foods). Finally, the sixth section assesses an individual’s perceived level of control over eating a craved food. 20-items are assessed by 100-mm visual analogue scales (VAS) and one item allows participants to enter their own response (*“Which one food makes it most difficult for you to control eating?”*).

In order to explore the underlying components of the COEQ, a principal component analysis (PCA) was conducted with an oblique rotation (direct oblimin) using 180 participants that had completed the scale in previous studies. The statistical details of the PCA can be found in Appendix 1. The PCA revealed five factors; Craving Intensity (5 items;  $\alpha = .88$ ), Positive Mood (4 items;  $\alpha = .76$ ), Craving for Sweet Foods (5 items;  $\alpha = .74$ ), Craving for Savoury Foods (4 items;  $\alpha = .69$ ), and Fullness (1 item). These factors were used in Chapters 7 and Chapter 8 as a measure of craving for food.

### **4.3 Laboratory food intake assessment**

All laboratory food intake assessment took place in individual experimental cubicles within the Human Appetite Research Unit (HARU) in the Institute of Psychological Sciences, University of Leeds. The HARU is a specially designed research facility that allows for the assessment of food intake in a controlled environment that is free from the extraneous variables present in the participant's natural environment. Within the experimental cubicles participants were isolated as much as possible from confounding variables that may have impacted their intake behaviour. These included isolation from variables such as extraneous smells and sounds, competing activities and any social stimuli. Additionally, in Chapter 8, the inter-meal interval was kept constant as previous research has shown that knowledge about the time until the next eating occasion impacts how much food is eaten, with individuals consuming more energy when the inter-meal interval is longer (De Graaf, De Jong, & Lambers, 1999).

Assessing energy intake in a laboratory setting has many advantages as high levels of control can be achieved over experimental variables such as energy and nutrient intakes, which can be assessed with a high degree of precision and accuracy. However, laboratory intake assessments can also constrain the participant's behaviour due to the artificial environment. Therefore, there is a trade-off between precision and naturalness (Blundell et al., 2009). There are two forms of food intake assessment in the laboratory. The first is a measure of fixed intake where the amount of food to be consumed is determined by the researcher and the second allows the participant to determine their own food intake in response to experimental manipulations and other variables present at the time (Stubbs, Johnstone, O'Reilly, & Poppitt, 1998).

#### **4.3.1 Fixed energy test meals**

One approach to the assessment of food intake is to fix the test meal either by the volume or the energy content of the food given. Fixed energy test meals allow for the

composition of the foods to be manipulated and standardised across participants. One benefit of using a fixed energy test meal is that they allow for greater experimental control in designs where food intake is used as an independent variable, compared to an ad libitum test meal approach. Fixed energy test meals were used in Chapters 5, 6, 7 and 9 in the current thesis. However, fixed energy test meals are arguably not the best method of inducing satiation due to individual differences in energy requirements that are likely to occur between participants. Therefore, in Chapter 7 the fixed energy test meal was calibrated to provide each participant with 25% of their estimated daily energy requirements in order to allow for individual differences in energy needs. As this calibration was not part of the design of the experiment reported in Chapters 5 and 6, ad libitum energy intake was adjusted post-hoc for differing energy requirements.

#### **4.3.2 Ad libitum test meals**

A further assessment of food intake in the laboratory is where the researcher provides the food in an ad libitum amount, weighing it before and after consumption. A range of foods are often provided for participants to choose from which allows for the assessment of quantitative data with regards to the amount of food consumed, and assessment for qualitative data with regards to nutrients or sensory aspects of the foods chosen. Ad libitum test meals can be more naturalistic than fixed energy meals as the participant is able to control the amount they eat in a manner similar to everyday life. However, caution must be exercised when selecting the foods to be given in an ad libitum test meal as research has shown that factors such as variety, palatability and energy density can induce over- or under-eating (Blundell & Macdiarmid, 2006; Hetherington, Foster, Newman, Anderson & Norton, 2006; Raynor & Epstein, 2001; Rolls, Van Duijenvoorde, & Rolls, 1984). Additionally, care must be taken with regards to the portion size of the food items given as research has demonstrated that larger portion sizes lead to greater energy intake regardless of participant characteristics (e.g. gender, BMI, or Restraint score) or method of serving (Rolls, Morris, & Roe, 2002).

### **4.3.3 Ad libitum snack intake**

The Bogus Taste test (BTT) is often used as a measure of ad libitum snack intake (e.g. Guerrieri et al., 2007; Nijs et al., 2010). In the BTT, participants are presented with a number of pre-weighed palatable snack foods (usually between four and six items) in ad libitum quantities and asked to rate the items on a number of variables (e.g. “*How salty is the food?*”) using 100-mm VAS. These ratings, rather than actual intake, are presented as the outcome of the task and the participant is invited to consume as little or as much as they would like whilst being seemingly unaware that their food intake is being monitored. The remaining foods are weighed after consumption allowing for the measurement of snack intake. The studies in current thesis used an adapted form of the BTT in order to reduce the unknown variance associated with differences in participant assumptions about the true nature and objectives of the task. To increase the level of control and reliability, participants were told the objective of the task without deception or ambiguity. To this end, the following standardised instructions were used:

“Please taste and rate, using the visual analogue scales provided, each of the six food items. We are interested in which items you like and how much you want to eat of them. You may eat freely from any of the bowls, but please try each one so that you can complete the ratings.”

In the current thesis, the adapted BTT is referred to as the ad libitum eating task and is used in Chapters 5 through 9. In Chapter 8, a modified version of the ad libitum eating task was shown to correlate with habitual snack intake [ $r(34) = .446$ ,  $p < 0.001$ ] indicating that the ad libitum eating task has a degree of external validity.

## **4.4 Free living dietary assessment**

Measurements of habitual energy intake from food diaries or dietary records are often very high in ecological validity as the participant is free to perform their



normal behaviours in their natural environment. However, data extracted from these methods can be unreliable as they rely on the participant's ability to remember what they have consumed, and their willingness and motivation to truthfully report all food and beverage items eaten. Furthermore, evidence suggests that recording food intake may result in the individual consuming less than they normally would due to an increase in self-monitoring (Baker & Kirschenbaum, 1993; Goris, Westerterp-Plantenga, & Westerterp, 2000). Dietary recall procedures, such as the United States Department of Agriculture's Automated Multiple Pass Method (AMPM; Moshfegh et al., 2008) can reduce the impact of such issues.

#### **4.4.1 24-hr dietary recall**

The AMPM (Moshfegh et al., 2008) is one of the most frequently used dietary recall procedures. The AMPM consists of five stages that are outlined in Table 4.1. The procedure takes between 30-45 minutes and measuring cups, spoons and images of food portions are usually provided to aid with the estimation of portion size. The AMPM favourably compares to Food Frequency Questionnaires (FFQ), a closed-ended procedure during which information about food intake over a specified period of time is gathered using a checklist of food and beverage items. Blanton, Moshfegh, Baer, and Kretsch (2006) demonstrated that total energy intake assessed by the AMPM did not differ significantly from estimated energy expenditure measured using doubly labelled water, whereas the FFQ underestimated total energy intake by 28%. Moshfegh et al. (2008) extended these findings, reporting that normal weight participants tended to underreport their energy intake by less than 3%. However, they found that as BMI increased so did the likelihood of underreporting. To try and overcome underreporting in overweight and obese participants the authors suggest that it is worthwhile disguising the nature of the interview so they do not alter their energy intake over the 24-hour reporting period.

The AMPM has a very low respondent burden compared to traditional food diary techniques, and any impact of self-monitoring is greatly reduced. Furthermore, the

AMPM has higher ecological validity compared to laboratory energy intake procedures. However, the main limitation of the AMPM is the increased time and effort needed from the researcher to collect and input all the information collected. The AMPM was used in Chapter 8 as a measure of habitual energy intake over a 24-hr period.

Table 4.1 The five stages of the Automated Multiple Pass Method for 24-hour dietary recall.

Step	Procedure
Quick list	The participant freely recalls all of the food and beverage items they have consumed over the preceding 24-hr period without interruption from the researcher.
Forgotten foods	The researcher cues recall of nine frequently forgotten food categories, which include non-alcoholic and alcoholic beverages, fruit, cheeses and bread items.
Time and occasion	The reported food and beverage items are reviewed and each item is assigned to an eating occasion (e.g. breakfast, snack).
Detail and review	Detailed information is gathered about brand names, recipes, portion size, added items (including condiments and fats), source (homemade or pre-packaged) and location of consumption.
Final probe	The participant reviews the information and is given the chance to recall any foods they may have missed, or report any small items of food they may not have felt was worth reporting. Finally, they assess whether their reported food intake was more, less or typical of their habitual intake.

#### **4.5 Measurement of subjective appetite sensations**

There are a number of systems that have been devised in order to ask participants specific questions relating to their subjective appetite sensations. These include VAS and labelled magnitude scales (LMS). VAS allow judgements to be made along a horizontal line (usually 100-mm) anchored by subjective statements at each end. The participant is required to make a mark along the line to indicate the intensity of a subjective sensation at that point in time therefore allowing the sensation to be measurable and quantifiable. VAS are used in a broad range of research and are a well-established method to measure subjective appetite sensations and have been

shown to be sensitive to experimental manipulations and to correlate with energy intake (Stubbs et al., 2000).

Advantages of VAS are that they can be easily applied and unambiguously interpreted by both participants and researchers. The most common type of VAS is administered using a pen and paper, which is efficient and has low participant burden. The traditional pen and paper procedure is especially useful under tightly controlled laboratory conditions where the timing of the completion of the scales can be monitored. However, under free-living conditions, or in instances where participants are permitted to leave the laboratory, the pen and paper method becomes much less reliable as compliance tends to be low and the ratings may be completed at the incorrect time points. To overcome this issue, a hand-held Electronic Appetite Ratings System (EARS II, (HP iPAQ)) can be used. The EARS II incorporates the VAS system on a portable handheld computer and has previously been validated (Gibbons, Caudwell, Finlayson, King, & Blundell, 2011). One advantage of the EARS II over the pen and paper method is the ability to set an alarm that prompts the participant to complete the ratings at the correct time point. In addition, each entry is time and date stamped allowing for the researcher to check compliance with the study procedures. In the current thesis, pen and paper VAS were used to measure subjective appetite sensations in Chapters 5, 6, 7 and 9. In Chapter 8, where participants were free to leave the laboratory, the EARS II was used.

#### **4.5.1 Satiety quotient**

The satiety quotient (SQ) is a measure of the satiating effect of food on an individual. The SQ relates to the amount of food consumed to the ratings of hunger before and following a meal. The SQ has previously been validated in previous research (Green, Delargy et al., 1997; Drapeau, King et al., 2007). The SQ was used in Chapter 7 to assess differences in the satiating effect of an individually calibrated fixed energy test meal and in Chapter 8 to assess the satiating efficiency of breakfast across the morning and of the ad libitum lunch and dinner test meals.

The following formula was used to calculate SQ:

$$SQ \text{ (mm/kcal)} = \frac{(\text{rating before eating episode} - \text{rating after eating episode}) * 100}{\text{Energy intake of the food consumed}}$$

## **4.6 Measurement of food reward**

### **4.6.1 Image validation**

In order to create a representative set of food cues, an array of food images were validated in an online survey for use in the current thesis. 115 respondents rated each image on various characteristics using a 7-point Likert scale. Respondents assessed the images on their perceived pleasantness (“1 = Not at all pleasant, 7 = extremely pleasant”), taste (“1 = sweet, 4 = bland, 7 = savoury”), fat content (“1 = low fat, 7 = high fat”) and calorie content (“1 = low calorie, 7 = high calorie”). Food images were selected if they were 1) recognisable, 2) perceived as being pleasant (mean scores >4) and 3) were rated as being representative of their proposed category (high fat savoury, low fat savoury, high fat sweet or low fat sweet). The outcome of this survey can be found in Appendix 2, along with further details about the cut offs used and the actual macronutrient composition of the selected foods.

### **4.6.2 The Leeds Food Preference Questionnaire**

The Leeds Food Preference Questionnaire (LFPQ; Finlayson, King, & Blundell, 2008) provides a measure of explicit liking, explicit wanting, and implicit wanting for an array of photographic food stimuli that vary in their nutrient and sensory qualities. The LFPQ has been validated in a wide range of research (Finlayson et al., 2011; Griffioen-Roose et al., 2010; Verschoor, Finlayson, Blundell, Markus, & King, 2010). In the current thesis, the photographic food stimuli were categorised according to fat content (high or low) and taste (sweet or savoury). Table 4.2 shows the validated standard list of food images used in the LFPQ in the current thesis. In instances where participants reported low acceptance of the foods (determined during screening) there were a few additional images for each category that could be substituted in.

Table 4.2 Photographic food stimuli used in the Leeds Food Preference Questionnaire to assess explicit liking, explicit wanting and implicit wanting

Savoury		Sweet	
High fat	Low fat	High fat	Low fat
Garlic bread	Cucumber	Jam biscuits	Apple
Crisps	Bread roll	Doughnuts	Strawberries
Chips	Pilau rice	Chocolate fingers	Skittles
Peanuts	Potatoes	Chocolate	Marshmallows

*Note.* A full summary of macronutrient content and energy values for the foods depicted in the food stimuli can be found in Appendix 2.

#### **4.6.2.1 Measurement of explicit liking and wanting**

To measure explicit liking, food images are presented individually, in a randomised order and participants are required to rate “*How pleasant would it be to taste some of this food now?*” on 100-mm VAS. Explicit wanting is assessed in a similar manner; only participants are required to respond to “*How much do you want some of this food now?*” (see Figure 4.1).

#### **4.6.2.2 Measurement of implicit wanting**

Implicit wanting is assessed using a forced choice methodology in which the food images are paired so that every image from each of the four categories is compared to every other image over ninety-six trials. Participants are required to respond according to “*Which food do you most want to eat now?*” as quickly and as accurately as possible (see Figure 4.2). Reaction time is covertly measured in milliseconds for each choice made.

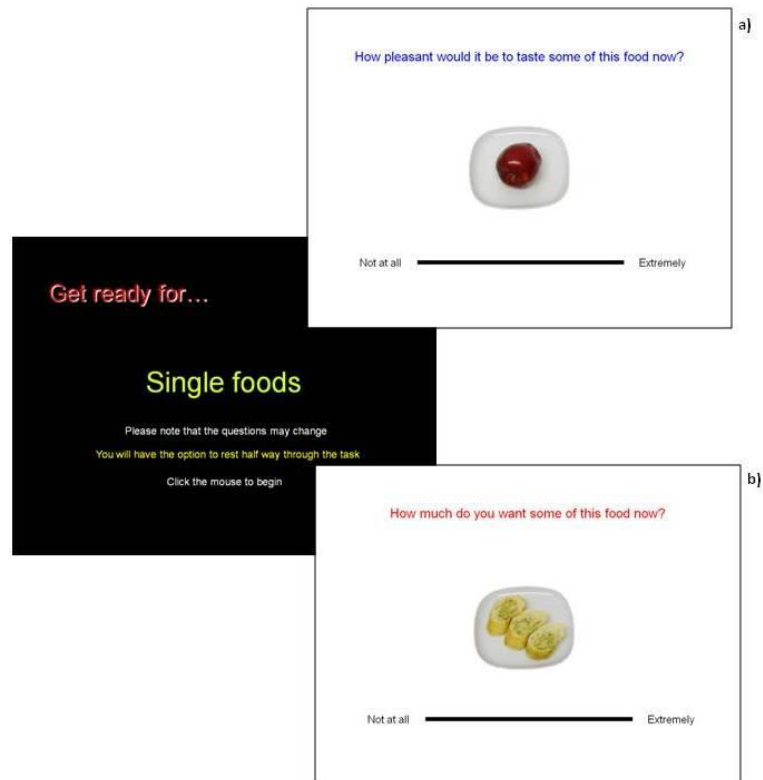


Figure 4.1 Representation of the explicit liking (a) and explicit wanting (b) trials in the LFPQ.



Figure 4.2 Representation of the implicit wanting trials in the LFPQ

#### 4.6.2.3 Scoring the LFPQ

Category scores are created according to the fat content and taste of the food images, resulting in four categories; high-fat savoury, low-fat savoury, high-fat sweet, and low-fat sweet. For explicit liking and explicit wanting, category scores are obtained by averaging the ratings for each category for each participant. A higher score indicates higher explicit liking or explicit wanting for that category. For implicit

wanting, reaction times are transformed to a standardised 'd-score' (D-RT) using a validated algorithm (Greenwald, Nosek, & Banaji, 2003). D-RT is calculated by 1) computing the overall standard deviation from pooled response trials; 2) computing the average reaction for each category; 3) computing the average reaction time for relevant comparison categories; 4) calculating the difference between category mean and comparison mean; 5) dividing by pooled standard deviation. D-RT is computed in order to improve statistical reliability and reduce contamination caused by individual variability in total average response speed. Scores are inverted for ease of interpretation so that a greater D-RT indicates a greater implicit wanting for one category relative to the other categories in the task. The LFPQ was used in all experimental chapters (5 through to 9) in the current thesis.

#### **4.6.3 The Visual Probe Task**

The modified Visual Probe Task (VPT; MacLeod et al., 1986) is a measure of attentional bias that assesses both the orientation and the maintenance of attention for visual food stimuli. The VPT has been used in a wide range of research examining attentional bias to reward related cues (Hepworth, Mogg, Brignell, & Bradley, 2010; Mogg, Bradley, Field, & De Houwer, 2003; Nijs et al., 2010). In the current thesis, the food stimuli used in the VPT were the same as those used in the LFPQ (see Table 4.2). An additional four images were included in order to match the number of images used in previous research. All food images were paired with neutral (office related) items to create twenty image pairs (see Appendix 3). The neutral items were matched as closely as possible with regards to shape, colour and position of the relevant food image. In conjunction with the LFPQ, food images were categorised according to fat content (high or low) and taste (sweet or savoury), so that there were five images in each category.

To assess the orientation and the maintenance of attention different exposure times are used. Exposure times of 100ms or 500ms are typically used to measure the orientation of attention whereas exposure times of 500ms or 2000ms are often used

to measure the maintenance of attention (Hepworth et al., 2010; Nijs et al., 2010). In the VPT, participants are presented with each image pair, the image pairs are then replaced by a probe (usually a dot or an arrow) for which participants are required to indicate either the location of the dot or the direction of the arrow using the keyboard as quickly and as accurately as possible while reaction time is measured (see Figure 4.3). To obtain an indicator of attentional bias size reaction times to the probe in the congruent trials are subtracted from those in the incongruent trials. A positive value indicates a greater attentional bias towards food. The VPT was used in Chapter 5.

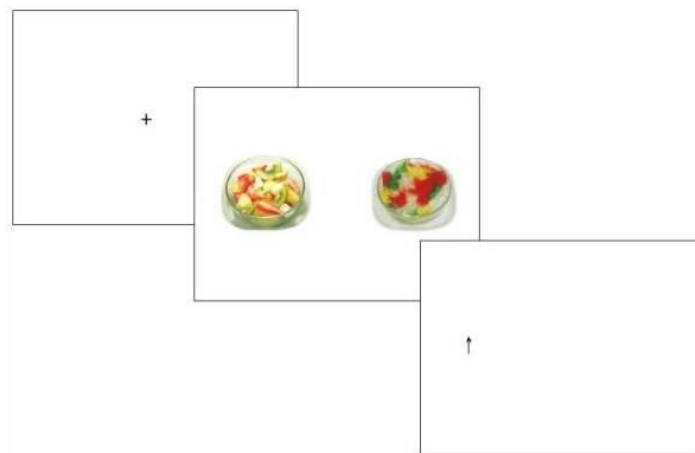


Figure 4.3 Representation of the Visual Probe Task.

## **4.7 Anthropometry and body composition**

### **4.7.1 Height, weight and waist circumference**

It is well evidenced that self-report measures of height and weight are often inaccurate, as individuals tend to over-estimate their height while under-estimating their weight often making self-report data unreliable (Palta, Prineas, Berman, & Hannan, 1982; Taylor et al., 2006). Self-reported height and weight can be especially problematic when identifying certain subgroups. For example, research suggests that overweight and obese individuals underreport their weight to a greater extent than normal weight individuals, and older adults tend to over-report their height more than younger adults (Dekkers, van Wier, Hendriksen, Twisk, & Van Mechelen, 2008; Larsen, Ouwers, Engels, Eisinga, & van Strien, 2008; Rowland, 1990). This makes it difficult to use self-reported height and weight to estimate body mass index for use as a categorical variable as the chances of misclassification would increase.



For this reason, in Chapters 5 through to Chapter 9, participants' height and weight were measured using a wall mounted stadiometer (KeWe, Germany) and scales to the nearest 0.1cm and 0.1kg, respectively under controlled conditions with participants wearing light clothing, and in all but one study following an overnight fast. In addition, waist circumference (in centimetres) was measured 1cm above the top of the participants' naval after expiration and was used as a measure of central adiposity. Waist circumference was measured three times to the nearest 0.1cm in order to obtain an average.

## **4.7.2 Body composition**

### **4.7.2.1 Bioelectrical impedance analysis**

Bioelectrical impedance analysis (BIA) is a relatively low cost and easy to use measure of body composition, and is one that is popular in clinics and in weight reduction programs. The procedure measures the resistance of an electrical current through the body tissue in order to calculate an estimate of total body water that is in turn used to provide an estimate of lean mass. Lean mass is then deducted from body weight to provide an estimate of body fat (Kyle et al., 2004). Research comparing the estimation of body fat by BIA with other measures is mixed. Sun et al. (2005) demonstrated that BIA is a good alternative for estimating body fat when participants are within a normal body fat range as BIA tends to provide an overestimation of percentage body fat when an individual's body fat is low, and an underestimation when an individual's body fat is high. However, Bolanowski and Nilsson (2001) reported a favourable comparison of BIA to dual-energy x-ray absorptiometry (DEXA) which suggested there were no differences in the measurement of lean mass, fat mass or percentage body fat. In the current thesis there was a strong correlation between BIA and air-displacement plethysmography estimates of body composition; fat mass [ $r(77) = .941, p < 0.001$ ], fat free mass [ $r(77) = .907, p < 0.001$ ] and percentage body fat [ $r(77) = .929, p < 0.001$ ]. BIA was used as an estimate of body composition in Chapter 5, 6, 7 and 9.

#### **4.7.2.2 Air-displacement plethysmography**

Air-displacement plethysmography (Bod Pod, Concord, CA) requires participants to sit in a sealed chamber wearing tight clothing and a swim cap to allow for an accurate measure of body volume. Body volume is assessed indirectly by measuring the volume of air a person displaces inside an enclosed chamber. Body volume is combined with measured body mass in order to calculate body density. Equations are then used to provide an estimate of body fat and fat free mass. A review of the literature suggests that the estimation of body fat from air-displacement plethysmography is within 1% to 2% similar to DEXA and hydrostatic weighing (Fields, Goran, & McCrory, 2002). Air-displacement plethysmography was used in Chapters 8 and 9 in the current thesis.

### **4.8 Genotyping**

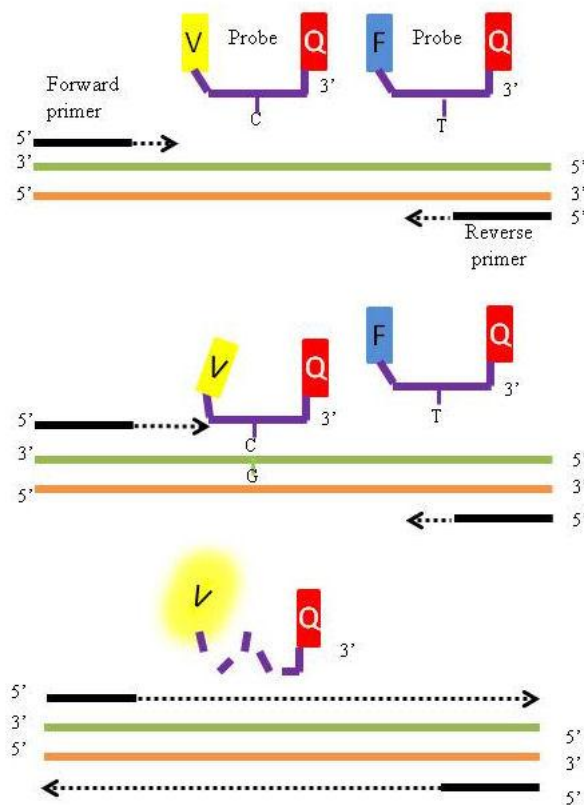
DNA for genotyping studies can be produced from virtually any tissue but most commonly DNA is extracted from blood or saliva samples. Research is mixed with regards to the quality and quantity of DNA derived from each sampling method. For example, Philibert, Zadorozhnyaya, Beach, and Brody (2008) suggest that the amount of DNA derived from saliva samples is much less than the amount of DNA derived from blood samples and that this impacts the genotyping success rate, 93.7% and 98%, respectively. In contrast, other researchers suggest that saliva samples provide good quality DNA that is comparable to DNA extracted from blood samples (Bahlo et al., 2010; Rylander-Rudqvist, Hakansson, Tybring, & Wolk, 2006). Using saliva samples rather than blood samples has a number of advantages. Firstly, unlike with a blood sample, providing a saliva sample is painless for the participant and secondly, the procedure does not require specialised skills.

One of the aims of the current thesis was to examine the association between common genetic variants and behaviours that may enhance susceptibility for overconsumption and weight gain using a candidate gene approach. In Chapter 9, seventeen single nucleotide polymorphisms (SNPs) in thirteen genes were isolated

from saliva and examined. DNA was extracted from saliva samples and genetic variants were analysed using the Taqman<sup>TM</sup> based approach (7500 Real Time PCR System, Applied Biosystems) and the Sequenom MassArray system (Sequenom, Inc).

#### **4.8.1 Taqman based approach**

The Taqman based approach utilises a dual labelled flourogenic probe that is complementary to the target sequence of DNA. The probe is an oligonucleotide with a fluorescent (reporter) and quencher dye attached to the 5' and 3' ends, respectively. The quencher dye, when the probe is intact, acts to decrease the fluorescent emitted by the reporter dye primarily by Förster-type energy transfer (Förster, 1948; Lakowicz & Maliwal, 1983). During the polymerase chain reaction (PCR) the flourogenic probe is annealed between the forward and reverse primer of the target sequence. During this process the reporter dye is separated from the quencher dye which increases the reporter dyes signal. The remaining probe is detached from the target strand so that the polymerase reaction may continue. This process is repeated during additional PCR cycles, resulting in a fluorescence intensity that is proportional to the quantity of amplicon produced (see Figure 4.4). To allow for allelic discrimination, the flourophores used in the current work were VIC<sup>®</sup> (4, 7,2'-trichloro-7'-phenyl-6-carboxyfluorescein) for allele 1 and FAM<sup>TM</sup> (6-carboxyfluorescein) for allele 2. Individuals were genotyped as homozygous for allele 1 when there was a substantial increase in VIC dye fluorescence only; in contrast those who were homozygous for allele 2 had a substantial in FAM dye fluorescence only. Heterozygote's were identified when both VIC and FAM fluorescence was increased. An advantage of the Taqman based approach is that it is less labour intensive compared to the Sequenom MassArray, as post-PCR processing is not required. However, a different probe needs to be synthesised (and purchased) for each SNP genotyped making it costly and time consuming when a large range of SNPs are analysed.



A) The fluorescent dyes emissions are inhibited when the probes are intact.

B) For each probe a fluorescent dye (V or F) and a quencher dye (Q) are attached at the 5' and 3' ends.

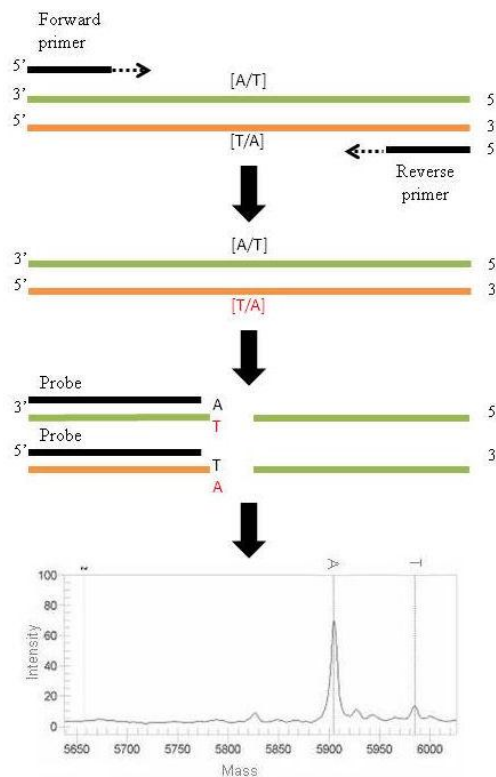
C) During polymerisation, the DNA polymerase cleaves the dye (V) from the complementary probe, separating it from the Q which results in increased fluorescence by V

Figure 4.4 Procedure for the Taqman based approach

#### 4.8.2 Sequenom MassArray

The Sequenom MassArray 4 analyser allows for the identification of up to 40 different SNPs in a single assay. The process begins with the custom design of a plex in which unique mass ranges are determined for each target SNP allowing for the identification of different allelic variants (see Figure 4.5). Following DNA amplification of the target SNPs by PCR, the DNA fragments are cycled through a single base extension (SBE) in which a single, mass modified, nucleotide is added to the extension probe. SBE allows for allelic variation at the sites of interest to be detected as different oligonucleotides are produced with different molecular weights that can be assessed using Matrix-Assisted Laser Desorption/Ionisation Time-Of-Flight (MALDI-TOF) mass spectrometry. The Sequenom MassArray is more cost effective than the Taqman as many target SNPs can be genotyped in one well.

However, the MassArray system is a complex multistage process that is susceptible to errors, which may result in the genotyping procedure being unsuccessful.



- A)** The target sequence(s) are amplified.
- B)** Enzymes are added to clean up superfluous nucleotides from the amplification process (SAP reaction).
- C)** A single unique mass modified nucleotide is added to the extension probe.
- D)** The different allelic variations have different molecular weights allowing them to be identified.

Figure 4.5 Procedure for the Sequenom MassArray

## 4.9 Statistical analyses

Data were analysed using Statistical Package for the Social Sciences v.20 (SPSS; IBM Corporation, Somers, New York). Data collected using E-Prime v.2.0 were exported to MS Excel via E-DataAid. Data from the online surveys were exported into MS Excel. MS Excel was used to calculate the variables for export to SPSS. All psychometric questionnaires and scales used were scored in accordance with the original authors instructions using MS Excel. Data were presented using the chart function of MS Excel by transferring the relevant descriptive statistics from SPSS rounded to two decimal places. All statistical procedures are described in greater detail in the method section of each experimental chapter.

## Chapter 5

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### **Comparing measures of food wanting: the Leeds Food Preference Questionnaire and the modified Visual Probe Task**

#### **5.1 Abstract**

The main aims of the current study were (1) to examine the association between two reaction time based behavioural tasks for the measurement of food-related motivation: the Visual Probe Task (VPT; orientation and maintenance of attentional bias) and the Leeds Food Preference Questionnaire (LFPQ; implicit wanting and explicit liking), (2) to examine how the VPT and the LFPQ perform under different motivational states and (3) to determine whether they can be used to predict the intake of palatable snack foods. Fifty-five normal weight females (age:  $21.38 \pm 2.9$ , BMI:  $22.09 \pm 2.37$ ) attended the research unit on three occasions. During the first two visits participants arrived in a fasted state and completed the LFPQ and the VPT under counterbalanced fasted or fed conditions. Ad libitum snack energy intake was assessed independently, in the third session. Findings from the VPT revealed an enhanced maintenance bias for food stimuli (compared to non-food control stimuli) in the fasted versus the fed condition. However, automatic orientation towards food stimuli was observed in both the fasted and the fed conditions. None of the outcome measures from the VPT were related to energy intake. The findings from the LFPQ were in accordance with previous research and showed that in the fed condition, ratings of explicit liking and wanting were lower for savoury foods, whereas implicit wanting was greater for sweet foods. Enhanced implicit and explicit wanting, and explicit liking for sweet foods were associated with greater overall energy intake and greater intake of sweet foods. Surprisingly, implicit wanting for low fat savoury foods was negatively associated with the intake of savoury snack foods. Finally, measures in the VPT and the LFPQ were not inter-correlated and may therefore assess different underlying processes. From these data it is concluded that the LFPQ is a more feasible, versatile measure of food-related motivation as it was more

sensitive to fasted and fed states, and was associated with measures of actual food choice and food intake. The LFPQ will be used to assess food reward in the remaining experimental chapters of this thesis.

## **5.2 Introduction**

The contribution and relationship between hedonic and homeostatic processes in the control of human appetite is a key issue in nutrition and obesity research today. While acute challenges to the homeostatic systems of appetite can often be adequately achieved by enforcing a period of fasting on participants in conjunction with long-standing appetite measures such as rating scales or test meal intake; the hedonic systems involved in appetite (liking and particularly wanting for food) have no such standard or widely-agreed metrics, and verifying their operation in human eating behaviour is controversial (Finlayson & Dalton, 2012a, 2012b; Havermans, 2012b). For a measure of food reward to be plausible it should incorporate the ability to not only reflect the existence of distinct components of reward, but also prevent confounding one component with another in order to allow for the detection of possible dissociations.

Liking for food appears to be a relatively stable characteristic within an individual and does not appear to be greatly modified by weight status (Cox, Perry, Moore, Vallis, & Mela, 1999). Liking is thought to be more important in determining the range of foods eaten (de Castro, Bellisle, & Dalix, 2000) and establishing the motivational value of food (Finlayson et al., 2008; Lowe & Levine, 2005). However, most research on food liking tends to use simple, often conspicuous self-report techniques that may be open to reporting bias, especially among individuals who are sensitive about their eating behaviour (Tooze et al., 2004). This is perhaps why the most recent methodologies to assess food reward have focussed on tasks that capture those aspects of reward that do not only rely on self-report and are less vulnerable to social desirability or presentation bias – such tasks are often interpreted as probing the food wanting component of reward. However, within the literature there have

been few attempts to directly compare existing experimental tasks to determine whether they produce similar outcomes under different experimental conditions, whether those outcomes correlate with each other, and therefore whether different tasks measure the same or different underlying components of reward.

Griffioen-Roose et al. (2010) conducted a study that employed two behavioural food reward tasks to measure the transfer effect of sensory specific satiety to foods from similar or dissimilar sensory domains. The authors used the Leeds Food Preference Questionnaire (LFPQ) and a progressive ratio food reinforcement task in separate experimental conditions following consumption of a predominantly savoury tasting or sweet tasting preload. A comparison of the tasks showed that both were able to capture sensory specific changes in wanting, and an asymmetric transfer between sweet and savoury foods to dissimilar foods. However, they concluded that the LFPQ task was more convenient for testing experimental hypotheses relating to food preferences and choice. Pothos, Calitri, Tapper, Brunstrom, and Rogers (2009) compared five measures of cognitive bias, including reaction time based measures of attentional bias (e.g. dot probe and emotional Stroop) and implicit attitudes (e.g. Extrinsic Affective Simon Task), to determine whether they assessed a single underlying process. Contrary to the authors' expectations, the relationships between the cognitive bias measures were very weak. However, the null result may be attributable to the nature of the design in which participants completed the five cognitive measures in succession, which may have resulted in testing fatigue. To reduce the likelihood of this, a better strategy would be to compare two behavioural tasks at a time.

The current study examined the relationship between two commonly used food reward tasks; the LFPQ (Finlayson et al., 2008) and the modified VPT (MacLeod et al., 1986). Both the LFPQ and the VPT record the reaction time of responses to food cues to provide a measure of motivation that can be interpreted as food wanting. In the VPT, attentional bias for food stimuli is determined by the hedonic value (or



motivational salience) of the food stimulus compared to a control image and this value can be modulated by internal motivational state. For example, a faster response in relation to food would be expected in a fasted compared to a fed state (Castellanos et al., 2009; Nijs et al., 2010). For the LFPQ, Finlayson et al. (2008) reported that satiation caused a decrease in explicit ratings of liking and wanting for a range of food stimuli varying in taste (sweet or savoury) and fat content (high or low). However, they found that participants' implicit wanting (as measured by the relative reaction time of forced-choice responses) increased for sweet foods but not for savoury foods in a fed state, suggesting that implicit wanting may be partly dissociable from explicit rating measures.

Taken together these findings suggest that the VPT and the LFPQ measures should differ according to fasted or fed states. Secondly, if the reaction time based outcomes of these tasks do reflect a similar motivational process – i.e. food wanting – they should be found to correlate in response to the same visual food stimuli.

### **5.2.1 Study aims**

There were three aims for the current study. The first was to examine how the VPT and the LFPQ perform under fasted or fed motivational states. The second aim was to determine whether the outcome measures on the two tasks were associated with the intake of palatable snack foods. The final aim was to investigate whether the VPT 'attentional bias' measure was related to the LFPQ 'implicit wanting' measure, to provide evidence for whether the tasks are measuring a shared underlying process – food wanting. Based on previous research, it was hypothesised that participants would have enhanced attentional bias for food in the fasted compared to the fed state. Additionally, it was hypothesised that participants would have enhanced implicit wanting for savoury foods when fasted compared to when they are fed.

## 5.3 Method

### 5.3.1 Participants

Fifty-five females (age:  $21.38 \pm 2.9$ , BMI  $22.09 \pm 2.37$ ) were recruited from the staff and student population at the University of Leeds. Participants were selected from an initial screening process to exclude those who were currently dieting, reported a history of eating disorders, or were unfamiliar with or disliked the study foods. Informed written consent was obtained prior to the study. All research procedures were reviewed and approved by the University of Leeds, Institute of Psychological Sciences Ethics Committee.

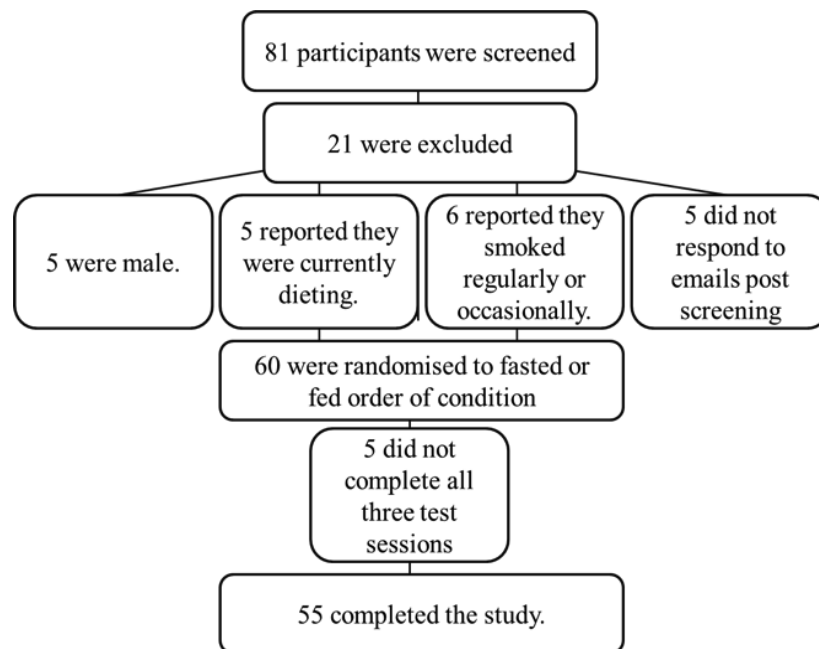


Figure 5.1 Outcome of the recruitment process; reasons for pre-study exclusion and attrition.

### 5.3.2 Design

The study conformed to a repeated measures design with participants attending the research unit on three occasions. The first two sessions were counterbalanced in a crossover design to minimise any potential order effects. Additionally, there was a seven-day minimum washout period between the first and the second session. The final session took place at least four days after session two. Participants were required to fast for three and a half hours prior to the commencement of sessions one and two and compliance with this instruction was assessed at the beginning of each

test session by questionnaire. Experimental task order (VPT and LFPQ) was counterbalanced across participants and experimental conditions. Prior to the first completion of the VPT participants were required to look through the control images in order to familiarise themselves with them and to avoid any confusion that may have occurred from mistaking the control items for food. In the final session, participants were requested to consume their normal lunch thirty minutes before the start of the test session so that they were in a fed state. Compliance with this instruction was checked by questionnaire at the start of the final test session.

### **5.3.3 Measures**

#### **5.3.3.1 Subjective Appetite Sensations**

Subjective ratings of appetite sensations were measured using 100-mm visual analogue scales (VAS). Measures of hunger (“How hungry do you feel right now?”) and fullness (“How full do you feel right now?”) were anchored at each end with the statements “extremely” and “not at all”. Ratings of prospective consumption (“How much food could you eat right now?”) and desire to eat (How strong is your desire to eat right now?”) were anchored at each end by “none at all” and “a very large amount” and “not very strong” and “very strong”, respectively.

#### **5.3.3.2 Food hedonics: explicit liking, explicit wanting and implicit wanting for food**

Explicit liking, explicit wanting and implicit wanting for food were assessed by the LFPQ, which is described in greater detail in Chapter 4. Food hedonics was assessed in a fasted and fed state.

#### **5.3.3.3 Attentional bias: orientation and maintenance of attention**

Attentional bias for food images was assessed using the modified VPT. All food images were paired with neutral (office related) items to create twenty image pairs that were categorised according to fat content (high or low) and taste (savoury or sweet) (see Appendix 3 for the image pairs). The neutral items were matched as

closely as possible with regards to shape, colour and position of the corresponding food image.

The VPT consisted of eight practice trials, followed by two blocks of 122 trials. Each block was preceded by two buffer trials, and consisted of eighty critical trials (food-neutral image pairs) and forty filler trials (neutral-neutral image pairs). Each trial started with a central fixation cross (500ms) followed by the appearance of an image pair displayed side by side for 100ms or 500ms to assess the orientation and maintenance of attention, respectively. An upwards or downwards facing arrow (the probe) directly followed the disappearance of the image pair, and appeared at the location of one of the images.

Participants were instructed to look attentively at the central fixation cross at the start of each trial, and to then identify the probe as quickly and as accurately as possible by pressing the ↑ key or ↓ key on the computer keyboard. If the participant did not respond, the probe disappeared after 5000ms. The inter-trial interval was 500ms. In half of the critical trials the probe appeared at the position of the food image (congruent trial), and half at the position of the neutral image (incongruent trial). Additionally, in half of the trials the probe was an up-arrow and in half the probe was a down-arrow. The filler trials were presented in a similar manner. The order of trials was randomised. In the critical trials image pairs were presented four times; twice on each side of the screen and twice in each congruence condition.

Participants' reaction time in identifying the probe was recorded in both the congruent and incongruent trials. In line with previous research (Castellanos et al., 2009; Nijs et al., 2010) reaction times of incorrect responses were excluded from the data. Outliers in reaction time were also excluded. Outliers were classified as responses that were less than 200ms, greater than 1500ms or exceeding the mean individual reaction time of the participant by plus or minus three standard deviations. Additionally, if a participant had responded incorrectly on more than 25% of the trials they were excluded from the analysis. A positive value was taken to indicate an

attentional bias towards food. Attentional bias for food was assessed in a fasted and fed state.

#### **5.3.3.4 Ad libitum eating task**

Participants were presented with six pre-weighed bowls of palatable high fat snack foods, chosen to be predominantly sweet (milk chocolate, chocolate finger biscuits and cookies) or savoury (ready salted crisps, salted peanuts and flavoured tortilla chips). The snacks were broken into irregular, bite size pieces (see Appendix 4 for an image of the ad libitum eating task layout and for nutritional information on the items used). Participants received 60 – 90g of each of the foods to ensure that the quantity of each food received looked the same. All of the bowls were presented at the same time and participants were asked to rate each food on a number of different properties including taste, blandness and saltiness using 100-mm VAS. Using standardised instructions, participants were informed that they could consume and rate the foods in any order and that which items they liked and how much they wanted to eat would be measured. In addition, they were told that they could consume as little or as much as they wanted as long as they at least tried each item. Foods were presented initially for ten minutes to allow for the ratings to be completed. The ratings were then taken away but the snack foods remained in the room whilst participants completed trait questionnaires. Food was measured to the nearest 0.1 g and energy values were determined using food tables and manufacturer labelling.

#### **5.3.3.5 Fixed energy test meal**

To create counterbalanced conditions of fasted and fed states, the study used a fixed energy test meal comprising of a cheese sandwich that provided participants with 25% of the 2000 calories the Government recommends for female daily energy requirements. The macronutrient content of the test meal was 23.1% CHO; 19.2% PRO; 57.7% fat. Participants ate alone in an experimental cubicle and foods were served at the same time across conditions. Participants were required to consume all

of the food that was provided to them. Food was measured to the nearest 0.1 g and energy values were determined using food tables and manufacturer labelling.

### 5.3.4 Procedure

Participants attended the Human Appetite Research Unit (HARU) on three occasions. The first two visits were conducted at lunchtime (between 12pm – 1:30pm) after participants had fasted for 3.5 hours following their usual breakfast (see Figure 5.2). In the fasted condition, participants completed the LFPQ and the VPT, followed by the fixed energy test meal. In the fed state condition, the procedures were identical except participants consumed the fixed energy test meal first and then completed the two tasks. Participants completed ratings of subjective appetite at the beginning of each test session, and after each event in the procedure. The final session was held after participants had consumed their own lunch. In this session participants were presented with the ad libitum eating task. Appetite ratings were taken before and after this eating task. Participants' height and weight were measured in light clothing at the end of the study procedures.

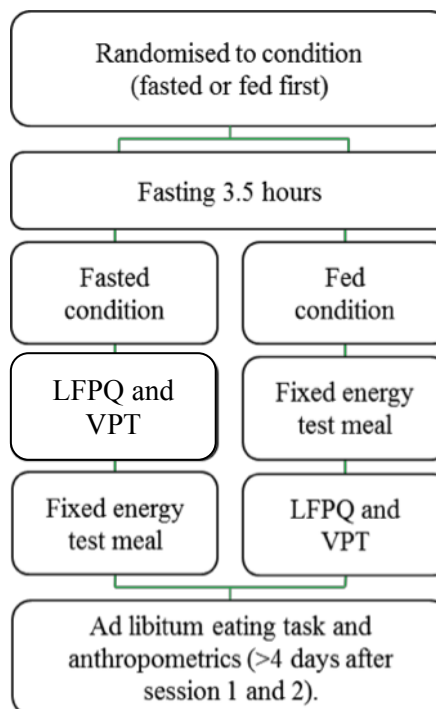


Figure 5.2 Study design.

### **5.3.5 Data Analysis**

Data were analysed using SPSS version 20 for Windows and are presented as means with standard deviations. The effect of condition (fasted or fed state) on appetite variables were assessed using a repeated measures analysis of variance (ANOVA). To assess the effect of condition on measures of overall (100ms and 500ms) attentional bias, paired samples t tests were used. In order to examine whether there were specific category biases, 2 (fasted or fed) x 2 (high fat or low fat) x 2 (sweet or savoury) repeated measures ANOVA were run for both 100ms and 500ms exposure times. 2 (fasted or fed) x 4 (high-fat savoury, low-fat savoury, high-fat sweet or low-fat sweet) repeated measures ANOVA were used to analyse the LFPQ variables. Pearson's correlations were run to assess whether variables on the VPT and the LFPQ were associated with energy intake from the ad libitum eating task and in order to determine whether the reaction time variables from both tasks were associated with one another. Where appropriate, Greenhouse-Geisser probability levels were used to adjust for non-sphericity. Post hoc analyses were conducted on significant interactions using the Bonferroni correction. An  $\alpha$ -level of 0.05 was used to determine statistical significance.

## **5.4 Results**

### **5.4.1 Subjective appetite sensations**

Appetite responses to the test meal were analysed to verify that participants underwent the measures in fasted and fed states. As expected, the test meal caused a significant decline in hunger and an increase in fullness, with participants reporting greater levels of hunger in the fasted condition compared to the fed condition [ $F(2, 108) = 138.85, p < 0.001$ ] and greater levels of fullness in the fed condition compared to the fasted condition [ $F(2, 108) = 84.52, p < 0.001$ ]. Ratings of desire to eat [ $F(2, 108) = 110.31, p < 0.001$ ] and prospective consumption [ $F(2, 108) = 93.28, p < 0.001$ ] also declined following the test meal.

## 5.4.2 The Visual Probe Task

### 5.4.2.1 Overall attentional bias

Table 5.1 shows the means and standard deviations of overall attentional bias from the 100ms trials and the 500ms trials for the fasted and fed conditions. In the fasted condition, participants showed attentional bias towards food for both 100ms trials and 500ms trials; although the mean was lower for the 500ms trials. When fed, participants showed attentional bias towards food in the 100ms trials but not in the 500ms trials. A paired samples t-test revealed that there was no significant effect of condition on attentional bias for 100ms trials [ $t(53) = -.105, p > 0.05$ ]. In the 500ms trials participants attentional bias for food was significantly lower when fed compared to when they were fasted [ $t(53) = 2.38, p < 0.02$ ].

Table 5.1 Mean (standard deviation) overall attentional bias scores for the 100ms and 500ms exposure times in the fasted and fed condition.

	Fasted	Fed
100ms	1.94 (20.5)	2.02 (14.88)
500ms	0.17 (12.79)*	-1.98 (11.5)*

\* $p < 0.05$

### 5.4.2.2 Attentional bias for specific food categories

#### 5.4.2.2.1 Orientation of attention: 100ms trials

There was no main effect of condition [ $F(1, 53) = .006, p > 0.05$ ], fat [ $F(1, 53) = 1.23, p > 0.05$ ] or taste [ $F(1, 53) = .902, p > 0.05$ ] on attentional bias scores. There was an interaction between condition and fat [ $F(1, 53) = 7.3, p < 0.01$ ]. Post hoc analyses revealed that attentional bias for high-fat foods was significantly greater in the fed compared to the fasted condition [ $p < 0.05$ ] (see Table 5.2).



Table 5.2 Mean (standard deviation) category attentional bias scores for the 100ms and 500ms exposure times in the fasted and fed condition.

	100ms		500ms	
	Fasted	Fed	Fasted	Fed
HFSA	-7.03 (29.74)	2.06 (29.1)	-1.07 (32.24)	-10.03 (32.82)
LFSA	5.27 (38.93)	1.25 (27.67)	2.61 (34.76)	-9.46 (31.63)
HFSW	-0.22 (36.01)	6.07 (29.96)	-3.9 (33.77)	-4.71 (30.5)
LFSW	8.86 (35.9)	-1.89 (30.64)	-0.75 (31.53)	9.87 (37.81)

#### 5.4.2.2.2 Maintenance of attention: 500ms trials

There was no main effect of condition [ $F(1, 53) = 1.11, p > 0.05$ ], fat [ $F(1, 53) = .094, p > 0.05$ ] or taste [ $F(1, 53) = 2.42, p > 0.05$ ] on attentional bias scores. There was an interaction between taste and condition [ $F(1, 53) = 5.51, p < 0.02$ ]. Post hoc analyses revealed attentional bias for savoury foods was significantly lower in the fed compared to the fasted condition [ $p < 0.05$ ] (see Table 5.2).

#### 5.4.2.3 Relationship to energy intake

As can be seen from Table 5.3, 100ms and 500ms overall and specific category attentional bias scores were not associated with energy intake in the ad libitum eating task.

Table 5.3 Pearson's correlations between energy intake and 100ms and 500ms attentional bias for the fasted and fed condition

	Fasted					Fed				
100ms	Overall	HFSW	HFSA	LFSW	LFSA	Overall	HFSW	HFSA	LFSW	LFSA
Overall energy intake	-.118	.069	-.014	-.048	-.234	.035	.038	-.111	.103	.029
Sweet energy intake	-.137	.067	-.063	-.097	-.190	.015	.058	-.115	.093	-.030
Savoury energy intake	-.054	-.038	.192	-.040	-.176	.079	.023	-.083	.013	.226
	Fasted					Fed				
500ms	Overall	HFSW	HFSA	LFSW	LFSA	Overall	HFSW	HFSA	LFSW	LFSA
Overall energy intake	.015	.064	.036	-.221	.130	.188	-.048	.099	-.018	.066
Sweet energy intake	-.025	-.058	.083	-.034	.026	.079	-.157	.002	.077	.049
Savoury energy intake	.000	.075	-.028	-.213	.071	.129	.008	.123	-.050	.007

### 5.4.3 Leeds Food Preference Questionnaire

#### 5.4.3.1 Explicit Liking

Ratings of explicit liking were significantly lower in the fed compared to the fasted condition [ $F(1, 50) = 28.62, p < 0.001$ ]. Bonferroni post hoc analysis of the main effect of category [ $F(3, 150) = 18.81, p < 0.001$ ] revealed that explicit liking for HFSW was greater than explicit liking ratings for all other categories whereas explicit liking was lowest for LFSA. A condition x category interaction was apparent [ $F(3, 150) = 26.12, p < 0.001$ ]. Post hoc analysis revealed that explicit liking ratings for savoury foods were significantly lower in the fed condition whereas there were no differences in explicit liking ratings for sweet items across conditions (see Table 5.4).

Table 5.4 Mean (standard deviation) explicit liking (mm) for the food categories in the fasted and fed condition

	Fasted	Fed
HFSW	62.01 (22.34)***	38.41 (22.39)***
LFSA	54.98 (18.12)***	37.85 (18.39)***
HFSW	69.06 (20.35)	65.12 (23.71)
LFSA	56.72 (17.06)	55.75 (18.77)

\*\*\* $p < 0.001$

#### 5.4.3.2 Explicit Wanting

Ratings of explicit wanting were significantly lower in the fed compared to the fasted condition [ $F(1, 50) = 30.84, p < 0.001$ ]. Bonferroni post hoc analysis of the main effect of category [ $F(3, 150) = 13.98, p < 0.001$ ] revealed that ratings of explicit wanting were highest for HFSW and lowest for LFSA. There was a condition x category interaction [ $F(3, 150) = 25.01, p < 0.001$ ]. Post hoc analysis revealed that explicit wanting ratings for savoury foods were significantly lower in the fed

condition whereas there were no differences in explicit wanting ratings for sweet items across conditions (see Table 5.5).

Table 5.5 Mean (standard deviation) explicit wanting (mm) for the food categories in the fasted and fed condition

	Fasted	Fed
HFSA	59.59 (24.37)***	34.19 (21.65)***
LFSA	54.32 (20.08)***	33.63 (17.58)***
HFSW	63.91 (21.47)	60.56 (26.38)
LFSW	53.32 (20.46)	52.81 (20.91)

\*\*\* $p < 0.001$

#### 5.4.3.3 Implicit wanting

In the fasted condition, participants responded faster for food items in the HFSA category. In the fed condition, participants responded faster for both sweet food categories compared to the savoury categories. A condition x category interaction [ $F(3, 144) = 13.35, p < 0.001$ ] was apparent. Post hoc analysis revealed that implicit wanting for HFSA was significantly lower in the fed compared to the fasted condition, whereas implicit wanting for LFSW was significantly higher (see Table 5.6).

Table 5.6 Mean (standard deviation) implicit wanting (D-RT) for the food categories in the fasted and fed condition

	Fasted	Fed
HFSA	.116 (.428)***	-.298 (.567)***
LFSA	-.010 (.418)	-.126 (.500)
HFSW	.043 (.399)	.171 (.478)
LFSW	-.124 (.543)***	.254 (.401)***

\*\*\* $p < 0.001$

#### 5.4.3.4 Relationship to energy intake

##### 5.4.3.4.1 Explicit liking

Explicit liking for HFSW was related to overall energy intake in both the fasted and the fed conditions. In the fed condition, overall energy intake was positively related to LFSW, and sweet food energy intake was positively related to explicit liking for HFSW (see Table 5.7).

Table 5.7 Pearson's correlations between energy intake (kcal) and explicit liking for the fasted and fed condition

	Fasted				Fed			
	HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Overall energy intake	.011	-.149	.295*	.117	.113	-.026	.373**	.245*
Sweet energy intake	-.073	.056	.188	.186	.118	.119	.274*	.198
Savoury energy intake	-.070	-.120	.003	-.094	.152	-.071	.177	.155

\* $p < 0.05$ ; \*\* $p < 0.01$

##### 5.4.3.4.2 Explicit wanting

Table 5.8 shows that overall energy intake was related to explicit wanting for HFSW in both the fasted and the fed condition. Additionally, overall energy intake was related to explicit wanting for LFSW in the fed condition. Additionally, in the fed condition, energy intake from sweet foods was positively related to explicit wanting for HFSW and LFSW.

Table 5.8 Pearson's correlations between energy intake and explicit wanting for the fasted and fed condition

	Fasted				Fed			
	HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Overall energy intake	.013	-.145	.354**	.233	.146	.091	.509***	.402**
Sweet energy intake	-.072	.006	.239	.197	.168	.210	.426**	.332*
Savoury energy intake	-.024	-.246	.086	.121	.111	.017	.245	.244

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

#### 5.4.3.4.3 Implicit wanting

As can be seen in Table 5.9, implicit wanting for HFSW was positively associated with overall energy intake and sweet food energy intake in both the fasted and fed condition, although the relationship was stronger in the fasted condition. There was also a negative relationship between implicit wanting for LFSA and overall energy intake and savoury food energy intake in both conditions.

Table 5.9 Pearson's correlations between energy intake and implicit wanting for the fasted and fed condition

	Fasted				Fed			
	HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Overall energy intake	-.159	-.425**	.453**	.153	-.098	-.298*	.337*	.076
Sweet energy intake	-.222	-.207	.340**	.096	-.097	-.167	.288*	.025
Savoury energy intake	-.054	-.392**	.189	.252	-.096	-.339*	.228	.223

\* $p < 0.05$ ; \*\* $p < 0.01$

#### **5.4.4 Task comparison: the Visual Probe and the Leeds Food Preference Questionnaire**

Pearson's correlations were conducted to examine the inter-relationship between the 100ms and 500ms exposure trials on the VPT and the implicit wanting measure on the LFPQ for the four food categories (see Table 5.10). There were no associations between the 100ms bias and implicit wanting in either condition. For the 500ms bias, there was a significant negative relationship between implicit wanting for HFSW and attentional bias for HFSA in the fasted condition. In addition, positive relationships were found in the fasted condition between implicit wanting for LFSW and attentional bias for LFSA, and implicit wanting for HFSW and attentional bias for LFSW. In the fed condition, there was a positive relationship between implicit wanting for LFSA and attentional bias for HFSW. There were no relationships between measures of attentional bias and explicit liking or explicit wanting (see Appendix 5).

Table 5.10 Pearson's correlations between 100ms and 500ms attentional bias scores and implicit wanting in the fasted and fed condition

		100ms				500ms			
Fasted		HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Implicit wanting	HFSA	-.144	.062	-.151	.202	.106	-.011	-.174	.068
	LFSA	-.079	.109	.028	-.097	-.044	-.024	.338*	-.183
	HFSW	-.060	.072	-.101	.035	-.380**	-.018	.085	.375**
	LFSW	.058	.101	-.066	-.142	.024	.329*	-.134	-.194
Fed		HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Implicit wanting	HFSA	.204	.111	-.221	-.161	-.238	-.046	.155	.200
	LFSA	-.011	.098	-.219	.084	-.263	-.059	.298*	.086
	HFSW	.196	-.025	-.027	-.158	.032	.006	-.147	.082
	LFSW	-.044	.124	-.043	.053	.081	-.015	-.099	.042

\* $p < 0.05$ ; \*\* $p < 0.01$

## 5.5 Discussion

The first aim of the current study was to ascertain whether the VPT and the LFPQ were sensitive to fasted or fed motivational states. Secondly, it aimed to determine whether responses on these tasks were related to palatable snack food intake. It was hypothesised that participants would have an enhanced attentional bias for food cues in the fasted condition compared to the fed condition. Additionally, it was hypothesised that participants would have an enhanced implicit wanting for savoury foods when fasted but that implicit wanting for these foods would decline in the fed state.

In accordance with the hypothesis, participants had enhanced attentional bias for food cues when fasted in both the 100ms and the 500ms conditions. However, attentional bias for food only declined in the fed state in the 500ms condition with a



slight increase evident in the 100ms condition. These findings are inconsistent with the findings from Nijs et al., (2010) who reported that when participants were fasted, they showed an enhanced automatic attentional bias towards food cues, which decreased following a test meal. Furthermore, Nijs et al. (2010) found no effect of motivational state on maintained attention, whereas in the current study there was an enhanced maintained bias for food cues when participants were fasted but not when they were fed. The disparity in findings may be partly attributable to differences in study design. For instance, the food stimuli used by Nijs et al., (2010) comprised solely of high calorie food images whereas in the current study the food stimuli varied, and were categorised, with regards to the fat content and the taste of the food. Using different categories of food images allowed for the assessment of specific food biases (which have not previously been reported in the literature), but this may have resulted in an overall attentional bias score that was not directly comparable to an overall attentional bias score for high calorie food images alone.

In the orientation of attention condition, examination of the specific food biases revealed that attentional bias for high fat foods was greater in the fed compared to the fasted condition. Furthermore, there appeared to be a trend indicating that in the fasted condition, participants had greater attentional bias for low fat foods. In the maintenance of attention condition, attentional bias for savoury foods was significantly lower in the fed compared to the fasted condition. In addition, in the fed condition there was a trend for a greater attentional bias for sweet foods, which appeared to be driven by the low-fat sweet food category. The latter finding is consistent with the literature on sensory specific satiation. For example, di Pellegrino, Magarelli, and Mengarelli (2010) decreased the hedonic value of a sweet or savoury palatable snack food by asking participants to eat them to comfortable fullness. They found that attentional bias decreased for the devalued snack food irrespective of its taste, whilst attentional bias for the uneaten snack food was not altered.

The findings from the LFPQ were consistent with previous research (Finlayson et al., 2008) as explicit ratings for liking and wanting for all food categories, especially the savoury categories, were lower in the fed compared to the fasted condition. The finding that explicit ratings were lower for all food categories in the fed state is in accordance with the notion that in the absence of internal need, food becomes less pleasurable as a consequence of it no longer being required to alleviate hunger (Cabanac, 1989). For the implicit wanting measure, participants responded faster to select high fat savoury food items in the fasted condition and faster for both high fat sweet and low fat sweet food items in the fed condition, with implicit wanting being greatest for the low fat sweet category. The findings indicate that the hedonic value of food is altered by motivational state, and may alter with regards to the type of food eaten. Indeed, the greater decrease seen in both the explicit ratings and the reaction time measure of wanting for savoury food items is most likely attributable to the savoury nature of the test meal and is in line with the literature on sensory specific satiation (Griffioen-Roose et al., 2010; Rolls et al., 1984).

The findings from the ad libitum eating task showed that the VPT did not relate to energy intake. For the LFPQ, all measures to some degree were related to energy intake – particularly overall energy intake and intake from sweet foods. Notably, there was a negative association between implicit wanting for low fat savoury foods and overall intake and savoury food intake in the ad libitum eating task. It may be tentatively suggested that having a high implicit wanting for healthier foods is a marker indicating resistance to the excessive consumption of high fat snacks (such as those used in the present study) and supports the notion that increased implicit wanting for food per se is not always a risk factor for overconsumption. Furthermore, it highlights the importance of including both high fat and low fat foods in the assessment of food hedonics. Additionally, this relationship was only observed in the more covert measure of implicit wanting and was not apparent in the explicit ratings.

The final aim of the current study was to assess whether the 100ms and 500ms attentional bias measures were related to implicit wanting in order to determine whether the two methodologies measured a shared underlying process – food wanting. One finding that was consistent between the two tasks was the greater implicit wanting and maintained attentional bias for low fat sweet foods in the fed condition. However, there were no other consistencies and no statistical associations between measures of attentional bias and implicit wanting for food and therefore the two tasks appear to assess different types of food wanting.

One possible explanation as to why the tasks were not related may be due to methodological differences in the tasks. The LFPQ presents participants with a series of food choices from which they must select which item they most want to eat at that moment in time. The ease with which this choice is made (reflected in the relative category response times) is dependent on the salience or the hedonic value of the food cues which will be in part mediated by current internal motivational state and individual preferences. The VPT on the other hand assesses the ‘attention grabbing’ properties of food images, which may be more distal to the behaviour of food intake than implicit wanting, which was developed to reflect the motivational process behind non-verbal food choice. Therefore automatic attentional bias may be a more elusive component of food-related motivation contained within implicit wanting, rather than a strong, independent determinant of actual food intake behaviour. However, due to the implicit wanting measure reflecting a more complex process, the two were not statistically related.

The current study had some limitations that need to be considered. First, the reliability of the VPT 500ms maintenance trials is questionable as participants may have utilised a task-related strategy in which following the initial orientation of attention towards either the food or control image, focus may have been switched back to the central position in order to prepare for the onset of the probe, or participants may have been in the process of switching between the two images at

probe onset which may have impacted their response times and therefore the outcome may not truly reflect where their attention had been predominantly focused. Using the VPT alongside more continuous measures of attentional bias such as eye-tracking techniques has been proposed to reduce the impact of this (Castellanos et al., 2009).

In conclusion, wanting as measured in the LFPQ is arguably a more sensitive, feasible and flexible measure of food wanting than the attentional bias measures of the VPT. This is supported by these data showing it is more responsive to a manipulation of motivational state and is meaningfully associated with measures of actual food choice and food intake. Therefore, the LFPQ will be carried forward as a tool for use in subsequent studies in this thesis.

## **5.6 Summary**

- Findings from the VPT revealed an enhanced maintenance bias for food stimuli compared to non-food stimuli in the fasted versus the fed condition. However, contrary to the hypothesis, automatic orientation towards food stimuli was observed in both the fasted and the fed condition.
- Neither the maintenance nor the orientation measures of attentional bias were related to palatable snack food intake in the ad libitum eating task.
- Measures of explicit liking, explicit wanting and implicit wanting in the LFPQ were not related to the attentional bias measures in the VPT. Therefore, the LFPQ and the VPT may assess different types of food wanting.
- Enhanced implicit and explicit wanting, and explicit liking for sweet food was associated with greater overall energy intake and greater intake of sweet foods in the ad libitum eating task.
- Overall, the current study suggests that the implicit wanting measure of the LFPQ is an arguably more sensitive, feasible and flexible measure of food wanting than the attentional bias measures of the VPT. Therefore, the LFPQ will be carried forward for the measurement of food hedonics in subsequent studies in this thesis.

## Chapter 6<sup>2</sup>

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### **Enhanced liking and wanting are markers for trait binge eating in normal weight females**

#### **6.1 Abstract**

The current study examined the influence of trait binge eating on liking and wanting, food choice and food intake in a sample of healthy normal weight females. Participants were divided into one of two groups using a tertile split of scores on the Binge Eating Scale; Lean ‘binge type’ (L-B) and lean ‘non-binge type’ (L-NB). Participants attended the research unit on three occasions, during the first two visits participants arrived following a 3.5-hour fast and completed the LFPQ under counterbalanced fasted and fed conditions. Ad libitum snack intake, anthropometrics and body composition were assessed in the final session. L-B had overall ratings of explicit liking for food that were similar in the fasted and fed condition, whereas L-NB overall ratings of explicit liking were significantly lower in the fed compared to the fasted condition. In addition, L-B had enhanced implicit wanting for high-fat sweet foods in the fasted condition compared to L-NB. Further to this, L-B consumed more sweet foods in the ad libitum eating task compared to L-NB. These findings suggest that the tendency to binge eat in a lean, non-clinical population is characterised by greater implicit wanting for, and consumption of high-fat sweet foods. Therefore, it is suggested that trait binge eating is functional at relatively low to moderate levels and may be a psychobiological marker for susceptibility to overeating.

#### **6.2 Introduction**

The characteristics of the current ‘obesogenic’ environment provide few barriers to prevent repeated overconsumption of highly palatable, energy dense foods and such overconsumption is a major determinant of weight gain (Swinburn et al., 2009).

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<sup>2</sup> This chapter is based on the same experimental study that was presented in Chapter 5 and therefore uses the same participants.

Therefore, it is reasonable to suggest that the hedonic system of appetite control plays a primary role in eating behaviour, capable of overriding homeostatic processes and driving consumption beyond energy needs. However, it is apparent that there is large individual variability in the susceptibility to over consume with some individuals being more susceptible to reward-driven overconsumption than others (Finlayson & Dalton, 2012b).

Binge eating is typically characterised by excessive consumption of food that is not driven by hunger or energy need (Brownley et al., 2007) and is often accompanied by feelings of guilt and perceived loss of control over eating (Ricca et al., 2009). Research in overweight and obese individuals has shown that binge eating behaviour is associated with increased cravings for sweet foods (Greeno et al., 2000) and increased food consumption in ad libitum eating tasks (Geliebter et al., 2001; Goldfein et al., 1993; Latner et al., 2009; Yanovski et al., 1992). In accordance with this, researchers have focussed on Binge Eating Disorder (BED) as a ‘hedonic subtype’ of obesity (Davis & Carter, 2009; Davis, Levitan, Carter, et al., 2008) that is characterised by differences in biologically mediated liking and wanting for food (Davis et al., 2009; Davis et al., 2012).

Research suggests that the behaviours and cognitions that underlie severe binge eating (seen in BED) may be relevant to the general, normal weight population – only at much lower levels (Finlayson et al., 2011). Finlayson et al., (2011) demonstrated that females with higher scores on the Binge Eating Scale (BES; Gormally et al., 1982) had higher liking for all food items assessed and a greater implicit wanting for high-fat sweet foods, specifically, compared to those with lower scores on the BES. Additionally, the enhanced wanting for sweet foods seen the high scorers coincided with them consuming significantly more high fat sweet foods in an ad libitum test meal compared to low scorers. Therefore, individual differences in trait binge eating may influence susceptibility for reward-driven overconsumption,

and may serve as a marker for future weight gain in healthy, normal weight individuals.

### **6.2.1 Study aims**

The aim of the current study was to examine the influence of trait binge eating on liking and wanting, food choice and food intake in normal weight (BMI: 18.5-24.9) healthy females. A secondary objective was to examine differences in body composition and health markers underlying variation in trait binge eating. It was hypothesised that higher trait binge eating scores would be associated with greater liking and wanting for food and greater intake of sweet snack foods in an ad libitum eating task.

## **6.3 Method<sup>3</sup>**

### **6.3.1 Participants**

To assess the effect of trait binge eating on food intake the sample was divided into high and low trait binge eating groups using a tertile split of scores on the Binge Eating Scale (BES; Gormally et al., 1982), individuals scoring  $\geq 12$  were categorised as lean ‘binge types’ (L-B), and those scoring  $\leq 7$  were categorised as lean ‘non-binge types’ (L-NB) (Figure 6.1).

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<sup>3</sup> The participants, methods and procedure for this chapter are the same as those outlined in Chapter 5 as the two chapters are from one experimental study. Therefore, to avoid repeating information, this section will only contain details specific to this chapter, however the procedure and study design figure have been included for convenience.

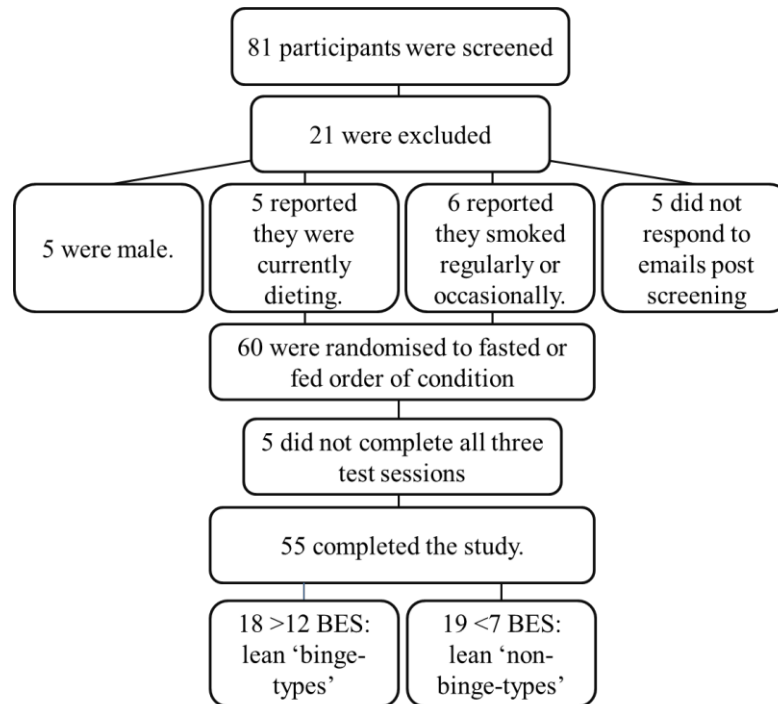


Figure 6.1 Outcome of the recruitment process; reasons for pre-study exclusion, attrition rate and categorisation into binge type groups.

## 6.3.2 Measures

### 6.3.2.1 Psychometric questionnaires

The Binge Eating Scale (BES; Gormally et al., 1982) and the Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick., 1985) were administered to participants at the end of the study procedures in order to assess trait binge eating and levels of restraint, disinhibition and hunger.

### 6.3.2.2 Body composition

Bioelectrical impedance (model BC418MA; Tanita Europe B.V., UK) was used in order to obtain an estimate of participants' fat mass, lean mass and percentage body fat. Participants were asked to remove any items from their pockets, to take off any heavy items of clothing and to remove their shoes and socks before body composition was assessed.



### 6.3.3 Procedure

Participants attended the Human Appetite Research Unit (HARU) on three occasions. The first two visits were conducted at lunchtime (between 12pm – 1:30pm) after participants had fasted for 3.5 hours following their usual breakfast (see Figure 6.2). In the fasted condition, participants completed the LFPQ, followed by the fixed energy test meal. In the fed condition, participants consumed the fixed energy test meal first and then completed the LFPQ. Participants completed ratings of subjective appetite at the beginning of each test session, and after each event in the procedure. The final session was held at least four days following sessions 1 and 2 and participants arrived within 30 minutes of having consumed their own lunch. In this session, participants were presented with the ad libitum eating task. Appetite ratings were taken before and after the ad libitum eating task. At the end of the study procedures participants completed the psychometric questionnaires and their height, weight and body composition were measured in light clothing.

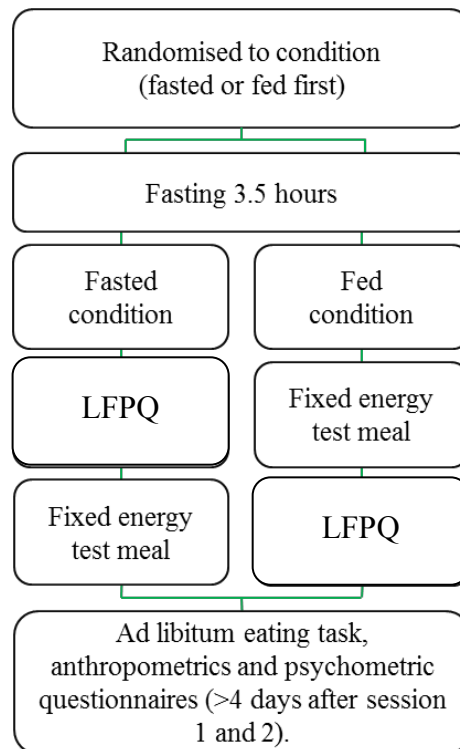


Figure 6.2 Study Design.

### **6.3.4 Data Analysis**

Data were analysed using SPSS version 20 for Windows and are presented as means with standard deviations. To examine the influence of trait binge eating on appetite sensations, energy intake and LFPQ variables, ANCOVA were used with trait binge eating scores examined as a covariate. For analyses where significant interactions between outcome variables and trait binge eating were revealed, these effects were further examined by dividing participants using a tertile split of scores on the BES (see Table 6.1). Where appropriate, Greenhouse-Geisser probability levels were used to adjust for non-sphericity. Post hoc analyses were conducted on significant interactions using the Bonferroni correction. An  $\alpha$ -level of 0.05 was used to determine statistical significance.

## **6.4 Results<sup>4</sup>**

### **6.4.1 Effect of trait binge eating on food intake and appetite variables**

As expected binge eating score was higher in L-B than L-NB [ $t(35) = 10.48$ ,  $p < 0.001$ ]. Additionally, L-B scored higher in trait disinhibition [ $t(35) = 4.54$ ,  $p < 0.001$ ] and trait hunger [ $t(35) = 2.69$ ,  $p < 0.01$ ] compared to L-NB and the difference in trait restraint approached significance [ $t(35) = 1.98$ ,  $p = 0.06$ ]. L-B had a greater percentage body fat [ $t(35) = 2.03$ ,  $p < 0.05$ ] and a greater amount of fat mass [ $t(35) = 2.06$ ,  $p < 0.05$ ] compared to L-NB.

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<sup>4</sup>There was no effect of binge eating score on either the 100ms or the 500ms attentional bias scores from the Visual Probe Task – the outcome of these analyses can be found in Appendix 6.

Table 6.1 Mean (standard deviation) age, anthropometrics, body composition and psychometric trait characteristics for L-B and L-NB.

	L-B (n=19)	L-NB (n=18)
Age	20.42 (2.04)	21.89 (2.37)
Weight (kg)	62.38 (8.65)	58.34 (6.81)
BMI (kg/m <sup>2</sup> )	22.57 (2.40)	21.52 (2.04)
Fat mass (kg)	17.82 (5.04)*	14.87 (3.48)*
% Body fat	27.97 (4.69)*	25.23 (3.78)*
Fat free mass (kg)	44.81 (4.68)	43.48 (3.99)
Binge eating score	17.26 (4.60)***	5.56 (1.15)***
Restraint	9.95 (6.29) <sup>1</sup>	6.56 (4.41) <sup>1</sup>
Disinhibition	9.79 (2.27)***	5.72 (3.18)***
Hunger	8.84 (3.58)**	6.50 (2.12)**

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ ; <sup>1</sup> $p = 0.06$

#### 6.4.1.1 Subjective appetite sensations

There was no effect of BES on ratings of hunger, fullness, prospective consumption or desire to eat in the fasted or the fed condition [ $F(2, 100) = .308, p > 0.05$ ;  $F(2, 100) = .114, p > 0.05$ ;  $F(2, 100) = .025, p > 0.05$ ;  $F(2, 100) = 1.52, p > 0.05$ , respectively] In addition, there was no effect of BES on ratings of appetite before or after the ad libitum eating task in the final test session [hunger:  $F(1, 49) = .285, p > 0.05$ ; fullness:  $F(1, 49) = .202, p > 0.05$ ; prospective consumption:  $F(1, 49) = .549, p > 0.05$ ; desire to eat:  $F(1, 49) = .652, p > 0.05$ ].

#### 6.4.1.2 Food choice and intake

The influence of trait binge eating on food intake in the ad libitum eating task was analysed for overall energy intake and intake according to the taste of the food (sweet or savoury). There was an interaction between BES and taste [ $F(1, 52) = 6.06, p < 0.02$ ] but no effect of BES on overall energy intake [ $F(1, 52) = 1.53, p > 0.05$ ]. Figure 6.2 illustrates these findings according to high and low tertile BES scores and shows that while both L-B and L-NB consumed a similar amount of

savoury snack foods, L-B consumed approximately 40% more sweet snack foods than L-NB.

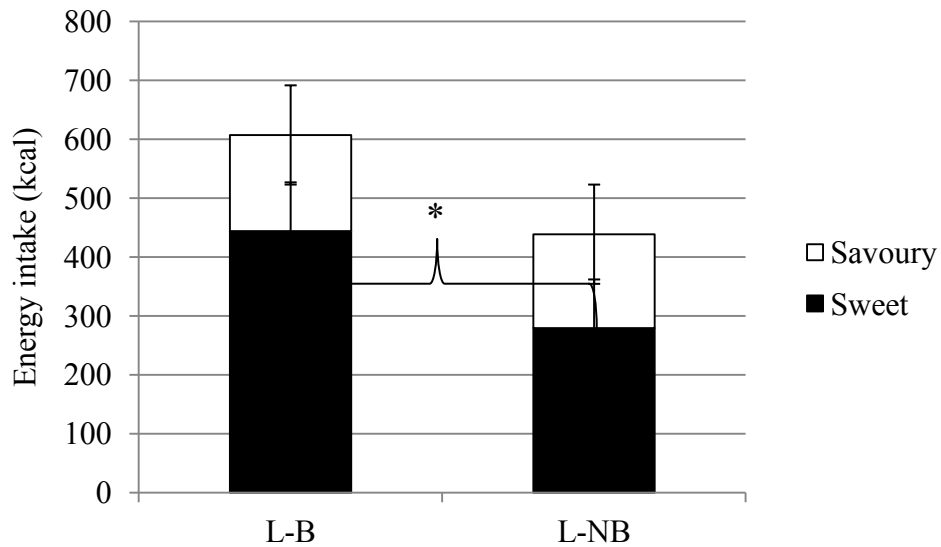


Figure 6.3 Energy intake (kcal) from the ad libitum intake task for L-B and L-NB

*\*p<0.05*

#### 6.4.2 Effect of trait binge eating on food hedonics

##### 6.4.2.1 Explicit liking

Table 6.2 shows that explicit liking ratings were similar for all food categories for L-B and L-NB in the fasted condition. In the fed condition, liking for HFSA, LFSA and HFSW was greater in L-B, whereas liking for LFSW was similar for L-B and L-NB. When the effect of trait binge eating on explicit liking was examined there was an interaction between condition and BES [ $F(1, 141) = 6.29, p < 0.02$ ]. Table 6.3 shows that overall liking for food was significantly lower in the fed compared with the fasted condition in L-NB, whereas overall liking ratings were similar in the fasted and fed condition for L-B.

Table 6.2 Mean (standard deviation) explicit liking ratings for L-B and L-NB in the fasted and fed condition

	Fasted		Fed	
	L-B (n=19)	L-NB (n=18)	L-B (n=19)	L-NB (n=18)
HFSA	61.99 (24.66)	63.13 (19.54)	51.38 (21.92)	32.35 (18.85)
LFSA	55.09 (17.54)	59.85 (17.86)	44.78 (14.81)	34.71 (18.34)
HFSW	64.74 (19.99)	65.32 (17.18)	68.76 (20.08)	66.94 (25.19)
LFSW	54.03 (18.44)	59.74 (17.33)	58.00 (16.54)	58.15 (21.51)

Table 6.3 Mean (standard deviation) overall explicit liking ratings for L-B and L-NB

	Fasted	Fed
L-B	58.96 (15.39)	55.73 (12.94)
L-NB	64.51 (9.61)*	48.04 (16.04)*

\* $p < 0.05$

#### 6.4.2.2 Explicit wanting

There was no effect of BES on ratings of explicit wanting (see Table 6.4).

Table 6.4 Mean (standard deviation) explicit wanting scores L-B and L-NB in the fasted and fed condition

	Fasted		Fed	
	L-B (n=19)	L-NB (n=18)	L-B (n=19)	L-NB (n=18)
HFSA	59.28 (24.53)	61.68 (22.37)	46.43 (22.07)	27.57 (19.39)
LFSA	55.45 (17.75)	60.03 (21.33)	40.61 (14.72)	30.91 (17.02)
HFSW	63.34 (19.52)	59.69 (21.00)	64.36 (19.45)	60.65 (32.58)
LFSW	50.66 (21.27)	58.74 (18.17)	52.65 (20.39)	55.66 (21.84)

### 6.4.2.3 Implicit wanting

There was a three-way interaction between condition, food category and BES [ $F(3, 138) = 4.84, p < 0.01$ ]. Post hoc examination revealed in the fasted condition, L-B had significantly greater implicit wanting for HFSW compared to L-NB, whereas L-NB significantly higher implicit wanting for LFSA. In the fed condition, L-NB responded faster than L-B for LFSW.

Table 6.5 Mean (standard deviation) implicit wanting (D-RT) scores for L-B and L-NB in the fasted and fed condition

	Fasted		Fed	
	L-B (n=19)	L-NB (n=18)	L-B (n=19)	L-NB (n=18)
HFSW	.161 (.344)	.218 (.447)	-.233 (.599)	-.291 (.624)
LFSA	-.172 (.427) <sup>a**</sup>	.091 (.385) <sup>a**</sup>	-.078 (.370)	-.307 (.581)
HFSW	.177 (.305) <sup>b**</sup>	-.148 (.482) <sup>b**</sup>	.191 (.450)	.158 (.572)
LFSW	-.164 (.542)	-.232 (.543)	.131 (.428) <sup>c*</sup>	.431 (.459) <sup>c*</sup>

\* $p < 0.05$ ; \*\* $p < 0.01$

## 6.5 Discussion

The current study examined the influence of trait binge eating on liking and wanting, food choice and energy intake in a sample of normal weight, healthy females. It was hypothesised that lean ‘binge-types’ (L-B) would have greater liking and wanting for food and consume more sweet snack foods in an ad libitum eating task compared to lean ‘non-binge types’ (L-NB).

Consistent with previous research (Finlayson et al., 2011), scores on the BES were associated with a greater preference for sweet foods in the ad libitum eating task, with L-B consuming approximately 40% more energy from sweet foods compared with L-NB. This finding complements previous research that has demonstrated the tendency to binge eat is often associated with increased cravings for sweet foods

(Greeno et al., 2000). Although the current study did not include a measure of food cravings, it would be advantageous to measure food cravings in the subsequent studies in order to determine whether trait binge eating is associated with increased cravings for sweet foods.

In addition to examining energy intake and food choice, the current study assessed whether trait binge eating was associated with differences in liking and wanting for food. It was hypothesised that L-B would have greater liking for food compared to L-NB. This hypothesis was partially supported, as while there were no differences between L-B and L-NB in liking for food in the fasted condition, in the fed condition liking for food was only significantly lower in L-NB. This perhaps suggests that L-B may have poorer appetite control as their explicit liking for food appeared, to a certain degree, resistant to a fed state as it was not significantly lower in the fed compared with the fasted condition, which was in contrast to L-NB and to previous research (Finlayson et al., 2008). Previous research has shown that greater liking for food is associated with susceptibility to weight gain (Blundell et al., 2005; Mela, 2001). Furthermore, this may also provide evidence of separation between the homeostatic and hedonic control systems of appetite in L-B as there were no differences in levels of hunger and fullness between L-B and L-NB. Therefore, in the fed condition, ratings of hunger and explicit liking for food were significantly reduced in L-NB, while only the former was significantly reduced in L-B.

The present study also hypothesised that L-B would have greater implicit wanting for food compared to L-NB. This hypothesis was supported as L-B had greater implicit wanting for high-fat sweet foods in both the fasted and the fed condition, whereas L-NB only responded faster for high-fat sweet foods in the fed condition. L-NB had greater implicit wanting for low-fat sweet foods in the fed condition suggesting that these individuals may have healthier automatic food preferences compared to L-B. The finding that L-B had a greater implicit wanting for sweet foods is in accordance with previous research (Finlayson et al., 2011). Interestingly, the positive

relationship between implicit wanting for high-fat sweet foods and BES was only present in the fasted state suggesting that the homeostatic and hedonic processes may combine to create a particularly vulnerable period in which the hedonic response to these types of foods is amplified which may have implications for appetite control. For example, Finlayson, Bordes, Griffioen-Roose, de Graaf, and Blundell (2012) examined the effect of a savoury or sweet tasting preload on energy intake in a sample of females who differed in their level of susceptibility to overeating (determined by their TFEQ disinhibition scores). They found that compared to the savoury tasting preload, energy intake in an ad libitum test meal increased following the sweet tasting preload in individuals scoring high in trait disinhibition.

When differences in body composition were examined, L-B had a greater percentage body fat compared to L-NB. Research examining binge-eating tendencies in children have also found higher levels of body fat in children who displayed eating disturbances (Tanofksy-Kraff, Yanovski, Wilfley, Marmarosh, Morgan, & Yanovski., 2004) and further to this the propensity to binge eat has been shown to predict fat mass gains during adolescence (Tanofsky-Kraff, Cohen, Yanovski, Cox, Theim, & Keil et al., 2006). This finding is of importance as previous research has shown that individuals categorised as 'normal weight' or 'lean' according to traditional BMI cut-offs may still be at risk for obesity-associated diseases (Gómez- Ambrosi et al., 2011). For example, Gómez-Ambrosi et al. (2011) reported that the risk for developing, and prevalence of Type-2 diabetes was greater in males and females with significantly higher percentage body fat even though they were BMI-categorised as lean.

The current study has some limitations that need to be considered. Firstly, it is important to note that the level of trait binge eating in the current sample only just reached moderate levels of severity (Marcus, Wing, & Hopkins, 1988). However, even at moderate severity, the present study demonstrated that trait binge eating was associated with strong, persistent liking for food, increased implicit wanting for high-



fat sweet foods, especially when fasted, and greater intake of these foods in an ad libitum eating task. Therefore, the findings of the current study suggest that trait binge eating is functional at low to moderate levels in a normal weight, non-clinical population and may therefore form part of a phenotype susceptible to overconsumption characterised by strong, persistent liking for food overall and enhanced implicit wanting for high-fat sweet foods specifically.

### **6.5.1 Limitations of Chapters 5 and 6**

The study presented in Chapters 5 and 6 have some limitations that need to be considered. Firstly, allowing participants to consume their own lunch outside of the laboratory may have impacted the outcome of the ad libitum eating task. For example, even though there were no differences in self-reported levels of hunger and fullness between L-B and L-NB, what they consumed for lunch may have affected their food choices and intake in the ad libitum eating task. Additionally, the ad libitum task would have benefitted from being counterbalanced so that participants were randomised to perform the task either before or after the main two experimental test sessions in order to control for any order effects that may have occurred from always having the ad libitum eating task at the end of the study. To address these limitations future studies in this thesis will include the ad libitum eating task in both the fasted and fed conditions. Furthermore, in order to control for a specific decrease in the hedonic value of savoury foods due to the savoury nature of the fixed energy test meal, subsequent studies in the current thesis will include both a savoury and a sweet component to the test meal.

## **6.6 Summary**

- Compared to L-NB, whose overall explicit liking for food was lower in the fed compared to the fasted condition, L-B overall explicit liking for food was similar in the fasted and fed condition.
- L-B had greater implicit wanting for high-fat sweet foods in the fasted condition compared to L-NB.

- While there were no differences in overall energy intake, L-B exhibited a greater preference for sweet snack foods in the ad libitum eating task, consuming approximately 40% more energy from them compared to L-NB.
- L-B had a greater percentage of body fat compared to L-NB.

**The effect of BMI and trait binge eating on liking and wanting for food**

**7.1 Abstract**

The current study examined the influence of trait binge eating in lean and overweight or obese females on liking and wanting, energy intake, and food choice. Using a matched pairs design, twenty-five lean and twenty-five overweight or obese females were categorised as either ‘binge-type’ or ‘non-binge type’ based on their scores on the Binge Eating Scale. Participants attended the research unit on three occasions, in the first session participants arrived following an overnight fast to have their height, weight and body composition assessed. For the final two sessions participants arrived to the research unit following a 3.5-hour fast and completed the LFPQ and an ad libitum eating task under counterbalanced fasted and fed conditions. Overweight or obese binge-types (O-B) consumed more energy in the ad libitum eating task than overweight or obese non-binge types (O-NB) and lean binge (L-B) and non-binge types (L-NB). O-B had greater liking for food overall and greater implicit wanting for high-fat sweet foods in both conditions compared to O-NB. In line with this, O-B reported greater food cravings for sweet foods. In the fasted condition, L-B had greater implicit wanting for LFSW compared to L-NB. These findings provide support for trait binge eating as a distinct ‘hedonic’ phenotype susceptible to overconsumption in overweight and obese females. This phenotype appears to be characterised by greater liking and wanting for food, increased energy intake and preference for high-fat sweet foods, and stronger experiences of food cravings. In addition, these findings suggest that explicit liking and implicit wanting may be useful markers for reward-driven overeating in this susceptible phenotype.

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<sup>5</sup> Parts of this chapter are based on a study that has been published “Dalton, M., Blundell, J. & Finlayson, G. (2013) Effect of BMI and binge eating on food reward and energy intake: further evidence for a binge eating subtype of obesity. *Obesity Facts*, **6**; 348-359.

## **7.2 Introduction**

Previous research has shown that individuals who are overweight or obese consume more energy in ad libitum eating tasks when compared to their lean counterparts, even when hunger is suppressed (Epstein, Temple, et al., 2007b; Nijs et al., 2010; Saelens & Epstein, 1996). Indeed, eating in a state of suppressed or absent hunger appears to be a key risk factor for overconsumption (Fisher & Birch, 2002). Therefore, it is important to consider the mechanisms of eating behaviour in the presence and absence of hunger. Furthermore, within the normal weight population several psychometric traits appear to reliably predict intake of highly palatable food across a range of experimental contexts (Finlayson et al., 2011; Guerrieri et al., 2007; Westenhoefer, Broeckmann, Münch, & Pudel, 1994) and this relative overconsumption often appears to be independent of any corresponding differences in sensations of appetite (Bryant et al., 2008). Reward driven eating appears to be able to override the inhibitory effects of satiety signals allowing for intake to extend beyond energy needs (Berthoud & Morrison, 2008) and may therefore constitute a distinct route to weight gain and obesity (Blundell & Cooling, 2000).

It has been noted that patients with Binge Eating Disorder (BED) display patterns of behaviour under experimental conditions that resemble enhanced food wanting (Davis et al., 2009; Finlayson & Dalton, 2012b; Svaldi et al., 2010). For example, Davis et al (2009) examined genetic and psychological indicators of hedonic eating in obese individuals with and without BED. They found that individuals with BED had a greater frequency of the A2 allele of the rs1800497 polymorphism in the Taq1A gene and the G allele of the A118G polymorphism in the OPRM1 gene, both polymorphisms have previously been related to enhanced receptor functionality (Kroslak et al., 2007; Noble et al., 1991; Ritchie & Noble, 2003). From these findings, Davis et al. (2009) have suggested that BED forms a distinct, biologically based, ‘hedonic enhanced’ subtype of obesity. Further support for differences in food wanting was found in a study comparing obese individuals with and without BED

using electroencephalography (EEG) to examine differences in attentional allocation for high and low calorie food (Svaldi et al., 2010). It was shown that individuals with BED had larger long latency event related potentials (an index of attentional and motivational processing) towards high calorie food images, which is consistent strong attention orientation and increased food wanting for these food images, compared to those without BED (Svaldi et al., 2010).

In the recent publication of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) BED has been reassigned from its DSM-IV category of a provisional eating disorder requiring further study to formally recognised eating disorder. Its estimated prevalence in the general population is between 0.7-3.0% and is commonly co-morbid with overweight and obesity (Brownley et al., 2007; Kessler et al., 2013). However, recurrent episodes of binge eating are estimated to occur in 10-20% of obese and lean individuals (Berg, Frazier, & Sherr, 2009; Bruce & Wilfley, 1996; Spitzer et al., 1993; Striegel-Moore et al., 2009) and constitute a trait that can be assessed psychometrically and applied to the general population. Importantly, the tendency to binge eat, assessed by the Binge Eating Scale (BES; Gormally et al., 1982) occurs at varying levels of severity in lean, overweight, and obese individuals. Alongside previous research, the data presented in Chapter 6 demonstrated that trait binge eating appears to be functional at low to moderate levels in a lean female sample and may be a reliable biopsychological marker for enhanced susceptibility to reward driven overeating in a non-clinical population.

### **7.2.1 Study aims**

The aim of the current study was to investigate individual differences in liking and wanting for food, and in energy intake in relation to trait binge eating and body mass index in lean and overweight or obese individuals. It was hypothesised that obese 'binge-types' would consume more food and display higher liking and implicit wanting for high-fat sweet foods independent of their motivational state. Additionally, it was hypothesised that lean and obese binge-types would exhibit a

preference for, and a greater intake of high-fat sweet foods in the ad libitum eating task compared with the lean and obese non-binge types.

## 7.3 Method

### 7.3.1 Participants

Twenty-five overweight or obese (age:  $25.3 \pm 8.9$ , BMI:  $30.7 \pm 3.1$ ) and twenty-five lean (age:  $27.2 \pm 8.3$ , BMI:  $22 \pm 1.4$ ) females were recruited from the staff and student population at the University of Leeds. Participants were selected from an initial screening process to exclude those who were taking medication, currently dieting, reported a history of eating disorders, or were unfamiliar with or disliked the study foods. Participants with a BMI between 18.5 and 24.9 were classified as lean, and participants with a BMI between 27.5 and 35 were classified as overweight or obese. The groups were individually matched by age, with an overall age difference of no more than three years across each matched pair. Informed written consent was obtained prior to the study. Participants received £8 for their participation. All research procedures were reviewed and approved by the University of Leeds, Institute of Psychological Sciences.

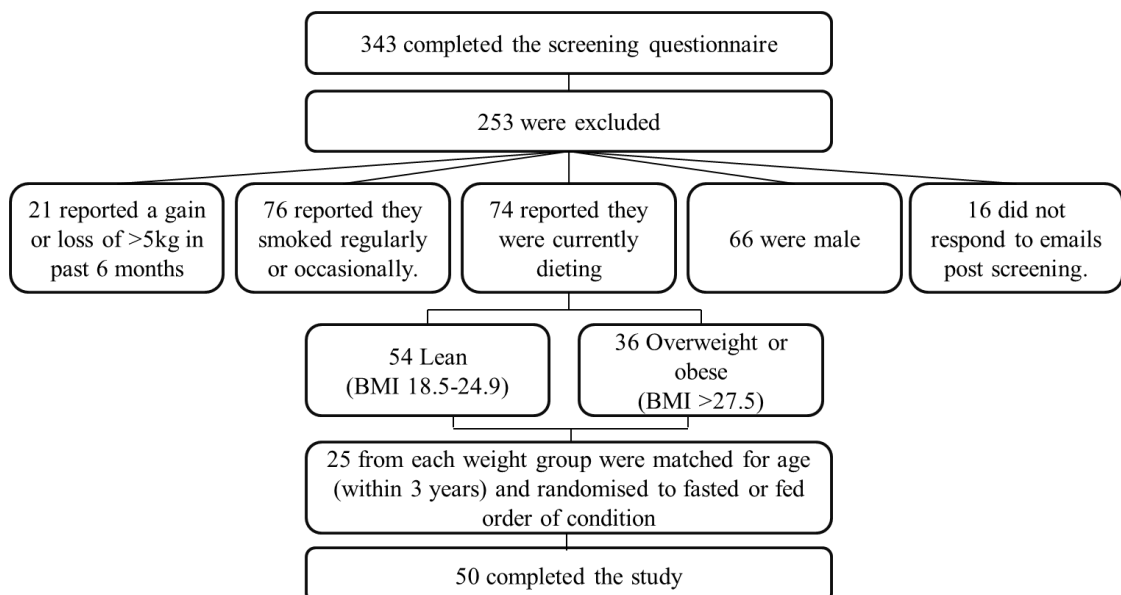


Figure 7.1 Outcome of the recruitment process; reasons for pre-study exclusion

### **7.3.2 Design**

The study conformed to a between subjects with repeated measures crossover design. Participants were randomised to completing the study procedures while in a fasted or a fed state over two test sessions separated by a minimum of seven days. Participants were asked to consume their normal breakfast, but to refrain from eating or drinking anything besides water for 3.5 hours before the start of the experiment. Participants were also required to attend one morning screening session for which they were required to fast from 10pm the evening before so that accurate measurements of height, weight, waist circumference and body composition could be taken. This session was held a minimum of seven days before the start of the experiment and allowed for the test meal to be calibrated according to individual energy requirements.

### **7.3.3 Measures**

#### **7.3.3.1 Subjective appetite sensations**

Measures of hunger, fullness, desire to eat and prospective consumption were taken using 100-mm VAS and are described in greater detail in Chapter 5.

#### **7.3.3.2 Food hedonics: explicit liking, explicit wanting and implicit wanting**

Measures of explicit liking, explicit wanting and implicit wanting were assessed using the LFPQ, which is described in greater detail in Chapter 4. Liking and wanting for food were assessed in the fasted and the fed condition.

#### **7.3.3.3 Psychometric questionnaires**

The Binge Eating Scale (BES; Gormally et al., 1982) and the Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick., 1985) were administered to participants at the end of the study procedures in order to assess binge eating severity, and levels of restraint, disinhibition and hunger. The Control of Eating Questionnaire (COEQ; Hill et al., 1991) was given to participants at the end of the study procedures in order

to assess their mood, appetite, and experience of food cravings over the previous seven days.

#### **7.3.3.4 Fixed energy test meal**

To create fasted and fed conditions, the current study used a fixed energy test meal containing a cheese sandwich and strawberry flavoured yoghurt. The test meal was individually calibrated to provide participants with 25% of their daily energy requirements (DER). Participants' DER was calculated using the Schofield equations (Schofield, 1985) for an estimate of basal metabolic rate, multiplied by physical activity level (PAL). PAL was assessed according to participants' self-report of the frequency and the type of exercise they engaged in per week. The macronutrient content of the test meal was 32% CHO; 21% PRO; 47% fat. Participants ate alone in an experimental cubicle and foods were served at the same time across conditions. Participants were required to consume all of the food that was provided to them. Food was measured to the nearest 0.1 g and energy values were determined using food tables and manufacturer labelling.

#### **7.3.3.5 Ad libitum eating task**

The ad libitum eating task was presented in a similar manner to what was described in Chapter 5 only this time it was presented in both the fasted and the fed condition. Additionally, there was an increase in the level of control over the amount of time participants were allowed to consume the foods with a time restriction of ten minutes. At the end of the ten minutes the snack foods and the VAS ratings were removed from the cubicle.

#### **7.3.3.6 Body composition**

Bioelectrical impedance (model BC418MA; Tanita Europe B.V., UK) was used in order to obtain an estimate of participants' fat mass, lean mass and percentage body fat. Participants were tested in the morning following an overnight fast and were



asked to remove any items from their pockets, to take off any heavy items of clothing and to remove their shoes and socks before body composition was assessed.

#### **7.3.4 Procedure**

Participants attended the research unit for one screening visit having fasted overnight, and two lunchtime visits having fasted for at least 3.5 hours following their normal breakfast (see Figure 7.2). During the screening visit participants' height, weight, waist circumference and body composition were measured. In the fasted condition, participants completed the LFPQ, followed by the ad libitum eating task and the fixed energy test meal. In the fed condition, the procedures were identical except that participants first consumed the fixed energy test meal after which a period of ten minutes passed to allow for the participants to feel full before the start of the LFPQ. After completion of the LFPQ, participants completed the ad libitum eating task. Subjective appetite ratings were measured at the beginning of each test session, and after each event in the procedure. On a separate day following their laboratory visits, participants completed a questionnaire booklet containing the psychometric questionnaires. They received written and verbal debriefing and were compensated for their time before leaving the study.

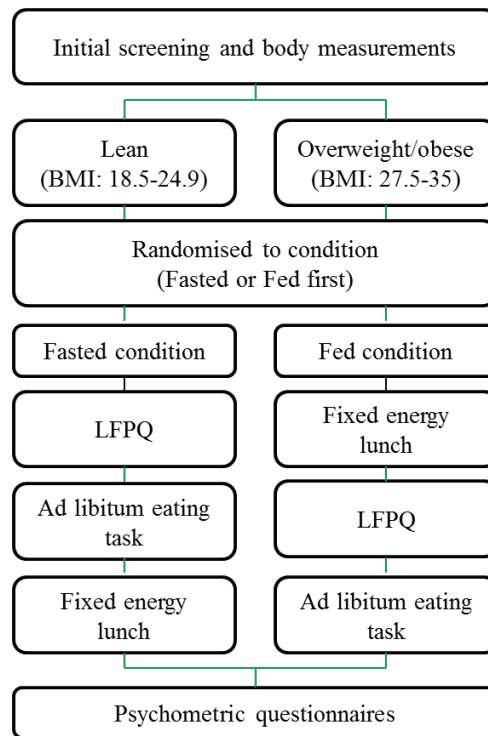


Figure 7.2 Study design

### 7.3.5 Data Analysis

Data were analysed using SPSS version 20 for Windows and are presented as means with standard deviations. The effect of condition (fasted or fed state) on appetite variables and the satiety quotient were assessed using repeated measures ANCOVA. To examine the influence of trait binge eating and BMI group on energy intake and LFPQ variables, ANCOVA were used with trait binge eating scores examined as a covariate. Therefore, overall energy consumed was examined according to condition and BMI group by a 2x2 ANCOVA. Food intake selection according to condition, taste (savory or sweet) and BMI group (lean or overweight/obese) was examined by a 2x2x2 ANCOVA and food hedonics according to condition, category (high-fat savory, low-fat savory, high-fat sweet and low-fat sweet) and BMI group were examined by a 2x2x4 ANCOVA. For analyses where significant interactions between outcome variables and trait binge eating were revealed, effects were further examined by dividing participants into four groups according to BMI group (lean and overweight or obese) and binge status (binge-type or non-binge type) following a median-split of scores on the BES.

## 7.4 Results

### 7.4.1 Sample characteristics

The final sample consisted of twenty-three overweight or obese and twenty-three lean individuals as four participants were excluded from the analyses as they later confirmed they did not comply with the study fasting procedures. Participant characteristics of age, BMI, body composition and psychometric traits are shown in Table 7.1.

Table 7.1 Mean (standard deviation) age, anthropometrics, body composition and psychometric trait characteristics of the lean and overweight or obese groups

	Lean	Overweight/obese
Age	27.22 (8.34)	25.35 (8.88)
BMI (kg/m <sup>2</sup> )	22.03 (1.43)***	30.73 (3.19)***
Fat mass (kg)	16.40 (3.79)***	34.32 (9.12)***
Body fat (%)	27.41 (4.68)***	39.89 (4.87)***
Lean mass (kg)	42.98 (2.72)***	50.25 (5.72)***
Waist (cm)	75.59 (5.57)***	98.96 (10.03)***
Restraint	8.17 (4.38)	8.57 (4.72)
Disinhibition	5.83 (3.46)**	9.65 (4.21)**
Hunger	5.65 (2.95)*	8.04 (4.05)*
Binge eating score	7.96 (6.57)**	15.52 (8.54)**

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

### 7.4.2 Subjective appetite sensations

Appetite responses to the test meal were analysed to check that participants underwent the procedures in fasted and fed states. The test meal caused a significant decrease in hunger, with participants reporting higher levels of hunger before the completion of the LFPQ and the ad libitum eating task in the fasted condition than in the fed condition [ $F(3, 135) = 65.15, p < 0.001$ ]. The results were also in the expected direction for fullness [ $p < 0.001$ ], desire to eat [ $p < 0.001$ ] and prospective consumption [ $p < 0.001$ ]. There were no significant differences between overweight or obese and

lean participants' self-reports of hunger [ $F(3, 132) = .444, p > 0.05$ ], fullness [ $F(3, 132) = .532, p > 0.05$ ], desire to eat [ $F(3, 132) = .047, p > 0.05$ ] or prospective consumption [ $F(3, 132) = .118, p > 0.05$ ].

#### **7.4.3 Ad libitum energy intake and food choice**

Participants consumed significantly more energy in the ad libitum eating task in the fasted condition (mean 444.47, SD 188.67) than in the fed condition (M: 349.23, SD 167.66; [ $t(45) = 4.39, p < 0.001$ ]). There was a main effect of food type on energy intake with participants consuming a greater proportion of sweet than savoury foods in both conditions ( $F(1, 45) = 23.59, p < 0.001$ ). Overweight or obese participants consumed more energy than lean participants [ $F(1, 44) = 4.70, p < 0.05$ ] in both the fasted (+24%) and the fed (+21%) condition. There were no differences in food choice (consuming sweet or savoury foods) between overweight or obese and lean participants (see Figure 7.3).

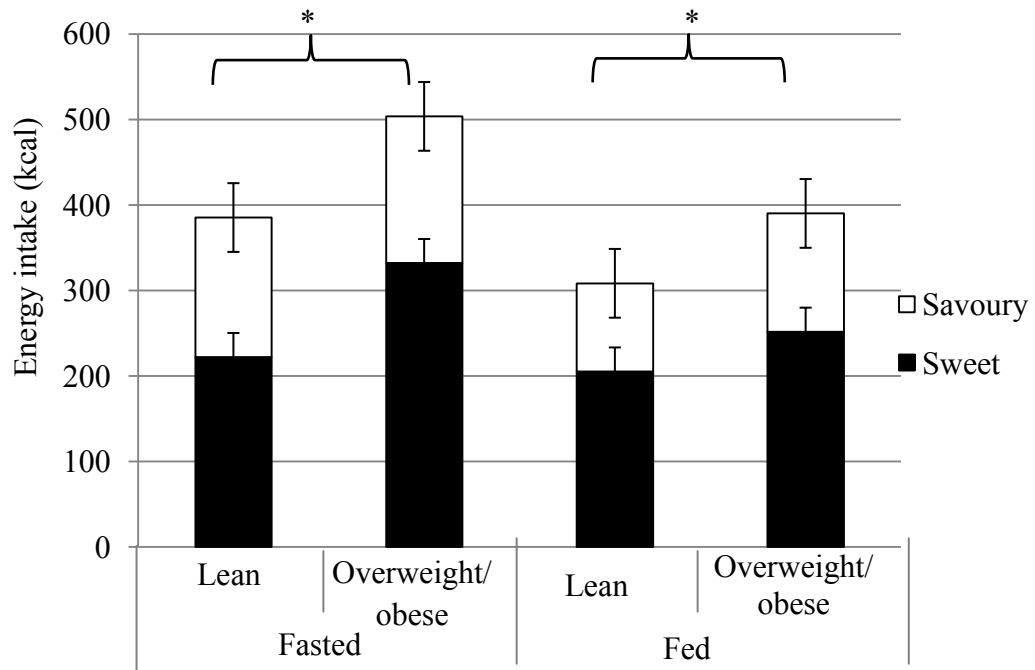


Figure 7.3 Energy intake from the ad libitum eating task for lean and overweight or obese participants in the fasted and fed condition

\* $p < 0.05$

#### 7.4.4 Effect of trait binge eating on energy intake, food choice and appetite variables

To assess the effect of trait binge eating on energy intake and food choice the lean and overweight or obese groups were categorised as either high or low in trait binge eating using a median split on the Binge Eating Scale. For the lean group, individuals with a score of  $\geq 6$  were categorised as lean 'binge type' (L-B) and those scoring  $\leq 5$  were categorised as lean 'non-binge type' (L-NB). For the overweight or obese group, individuals with a score of  $\geq 15$  were categorised as obese 'binge-type' (O-B) and those scoring  $\leq 14$  were categorised as obese 'non-binge type'. Table 7.2 summarises the characteristics of the newly created groups.

Table 7.2 Mean (standard deviation) age, anthropometrics, body composition and psychometric trait characteristics for O-B, O-NB, L-B and L-NB.

	O-B (n=11)	O-NB (n=12)
Age	25.82 (8.79)	25.36 (9.39)
BMI (kg/m <sup>2</sup> )	31.70 (4.10)	29.68 (2.11)
Fat mass (kg)	37.54 (10.27)	30.78 (6.40)
Body fat (%)	41.12 (5.46)	38.53 (3.97)
Lean mass (kg)	52.59 (4.59)	47.42 (5.55)
Waist (cm)	103.42 (10.54)*	94.53 (11.30)*
Restraint	9.67 (4.68)	7.36 (4.68)
Disinhibition	11.92 (3.55)**	7.18 (3.49)**
Hunger	10.25 (3.86)**	5.64 (2.73)**
Binge eating score	21.50 (7.04)***	9.00 (4.05)***
	L-B (n=12)	L-NB (n=11)
Age	23.3 (7.36)*	30.23 (8.01)*
BMI (kg/m <sup>2</sup> )	22.44 (1.47)	21.71 (1.36)
Fat mass (kg)	17.28 (3.88)	15.79 (3.75)
Body fat (%)	28.76 (4.60)	26.47 (4.68)
Lean mass (kg)	42.48 (2.46)	43.32 (2.94)
Waist (cm)	73.81 (3.98)	74.11 (4.93)
Restraint	9.10 (4.01)	7.46 (4.67)
Disinhibition	8.70 (2.26)***	3.62 (2.43)***
Hunger	6.30 (2.98)	5.15 (2.94)
Binge eating score	14.50 (4.71)***	2.92 (1.71)***

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$ .

Table 7.2 shows that O-B and L-B had higher trait binge eating and disinhibition scores than O-NB and L-NB, respectively. Additionally, O-B scored higher on trait hunger than O-NB. There were no differences in BMI, body composition or waist circumference between the two lean groups. Compared to O-NB, O-B had a larger waist circumference [ $t(21) = 2.098$ ,  $p < 0.05$ ]. There were no other differences between O-B and O-NB.

#### **7.4.4.1 Subjective appetite sensations**

There was no effect of BES on ratings of hunger, fullness, prospective consumption or desire to eat in the fasted and the fed condition [ $F(3, 132) = 1.06, p > 0.05$ ;  $F(3, 132) = 2.56, p > 0.05$ ;  $F(3, 132) = .827, p > 0.05$ ;  $F(3, 132) = .872, p > 0.05$ ], respectively.

#### **7.4.4.2 Satiety Quotient**

There was no effect of BES on the satiating efficiency of the fixed energy test meal in either the fasted [ $F(1, 44) = .062, p > 0.05$ ] or fed condition [ $F(1, 44) = 1.77, p > 0.05$ ].

#### **7.4.4.3 Energy intake and food choice**

The influence of trait binge eating on food intake in the ad libitum eating task was analysed for overall energy intake, and intake according to the taste (sweet or savoury) of the food (see Figure 7.4). For overall energy intake, there was a three-way interaction between condition, BES and BMI group [ $F(1, 42) = 10.16, p < 0.01$ ]. To further explore this interaction, the differences between the four groups were analysed. It was revealed that O-B consumed more energy overall in the fasted and fed condition compared to O-NB and both lean types [ $p < 0.01$ ] and this increase in energy intake appeared to be driven by increased consumption of sweet foods specifically. There were no significant differences in overall energy intake between O-NB, L-B and L-NB (Figure 7.4). For intake according to taste of the food, there was an interaction between taste and BES [ $F(1, 43) = 12.93, p < 0.001$ ] which demonstrated that greater binge eating scores in both lean and overweight or obese individuals was associated with a greater preference for sweet foods.

In summary, overweight or obese participants consumed more energy ad libitum compared to lean participants. However, when the influence of trait binge eating on energy intake and food choice was analysed, greater overall energy intake (by approximately 30%) was only observed in those individuals who were overweight or

obese and had higher binge eating scores. Higher binge eating scores were also associated with a greater preference for sweet foods in both lean and overweight or obese individuals.

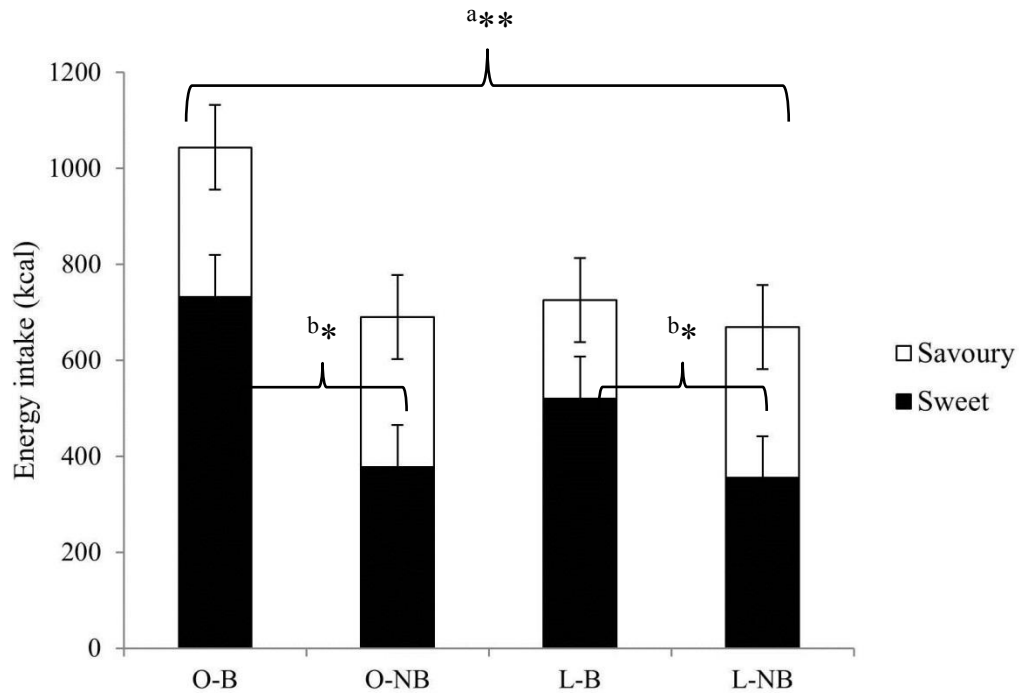


Figure 7.4 Energy intake and intake from sweet and savoury food items for O-B, O-NB, L-B and L-NB from the fasted and fed condition

\* $p < 0.05$ ; \*\* $p < 0.01$

<sup>a</sup>Condition  $\times$  BES  $\times$  BMI group

<sup>b</sup>Taste  $\times$  BES

#### 7.4.5 Effect of trait binge eating on cravings for food

Trait binge eating was positively associated with Craving for Sweet Food [ $r(42) = .432$ ,  $p < 0.01$ ] and Craving Intensity [ $r(42) = .534$ ,  $p < 0.001$ ], and negatively associated with Positive Mood [ $r(42) = -.361$ ,  $p < 0.05$ ] on the COEQ. When these relationships were explored further it was revealed that O-B scored significantly higher on the Craving Intensity [ $t(19) = 2.75$ ,  $p < 0.01$ ] and the Craving for Sweet Food [ $t(19) = 3.75$ ,  $p < 0.001$ ] subscales when compared to O-NB. In addition, O-B scored significantly lower on the Positive Mood subscale [ $t(19) = 2.18$ ,  $p < 0.05$ ]. There were no differences in cravings between the lean groups.



## 7.4.6 Effect of trait binge eating on food hedonics

### 7.4.6.1 Explicit liking

Table 7.3 shows that ratings of explicit liking for food were higher for all groups in the fasted compared to the fed condition [ $F(1, 42) = 46.07, p < 0.001$ ]. A between subjects effect revealed that O-B had a higher explicit liking for all foods compared to O-NB and the two lean groups [ $F(3, 42) = 3.23, p < 0.03$ ]. Additionally, there was an interaction between binge type and food type. Post hoc analyses revealed that O-B had greater explicit liking for both high-fat sweet and low-fat sweet foods compared with O-NB in the fasted condition [ $F(9, 126) = 2.24, p < 0.02$ ].

Table 7.3 Mean (standard deviation) explicit liking ratings (mm) for O-B, O-NB, L-B and L-NB for the food categories in the fasted and fed condition

Fasted	O-B (n=11)	O-NB (n=12)	L-B (n=12)	L-NB (n=11)
HFSA	68.56 (18.89)	51.64 (17.98)	49.25 (25.02)	64.50 (16.56)
LFSA	53.33 (19.23)	50.70 (16.47)	50.35 (25.93)	54.48 (21.17)
HFSW	71.94 (9.81) <sup>a*</sup>	52.02 (30.46) <sup>a*</sup>	53.70 (22.17)	46.44 (24.96)
LFSW	64.42 (10.02) <sup>b*</sup>	48.84 (22.82) <sup>b*</sup>	54.43 (15.79)	46.52 (20.64)
Fed	O-B (n=11)	O-NB (n=12)	L-B (n=12)	L-NB (n=11)
HFSA	44.67 (26.89)	33.61 (20.70)	23.03 (25.80)	30.04 (21.08)
LFSA	38.63 (21.71)	30.52 (18.98)	28.93 (18.55)	24.90 (21.43)
HFSW	60.19 (18.43) <sup>c*</sup>	35.09 (28.27) <sup>c*</sup>	50.93 (23.15)	42.73 (23.37)
LFSW	54.94 (16.97) <sup>d*</sup>	34.50 (22.00) <sup>d*</sup>	51.80 (23.19)	47.17 (20.20)

\* $p < 0.05$

Note: <sup>a-d</sup> indicate the significant post-hoc comparisons for the interaction between binge type and food type.

### 7.4.6.2 Explicit wanting

Table 7.4 shows that ratings of explicit wanting for food were higher in the fasted compared to the fed condition [ $F(1, 42) = 51.49, p < 0.001$ ]. A condition by food type interaction [ $F(3, 126) = 15.07, p < 0.001$ ] revealed that, overall, in the fasted

condition, participants' ratings of explicit wanting were higher for HFSA foods, and in the fed condition, participants ratings' were higher for both sweet food categories. Finally, there was an interaction between binge type, food type and condition [ $F(9, 126) = 2.89, p < 0.01$ ]. Post hoc analyses revealed that for O-B had greater explicit wanting for sweet foods in the fed condition compared to O-NB, whose ratings of explicit wanting were significantly lower for all food categories in the fed compared to the fasted condition.

Table 7.4 Mean (standard deviation) explicit wanting ratings (mm) for O-B, O-NB, L-B and L-NB for the food categories in the fasted and fed condition

Fasted	O-B (n=11)	O-NB (n=12)	L-B (n=12)	L-NB (n=11)
HFSA	63.16 (19.17)	54.63 (23.25)	44.73 (23.28)	63.79 (21.49)
LFSA	49.39 (18.84)	46.50 (16.75)	50.38 (24.23)	51.00 (20.26)
HFSW	68.89 (10.75)	49.52 (32.71)	49.38 (24.04)	41.69 (25.38)
LFSW	59.39 (9.57)	49.73 (24.90)	50.88 (13.77)	43.23 (21.32)
Fed	O-B (n=11)	O-NB (n=12)	L-B (n=12)	L-NB (n=11)
HFSA	37.07 (27.67)	32.38 (25.24)	20.68 (25.70)	28.50 (21.40)
LFSA	36.00 (19.41)	23.40 (19.15)	25.80 (18.79)	19.33 (17.95)
HFSW	55.46 (22.87) <sup>a*</sup>	32.13 (28.97) <sup>a*</sup>	42.93 (24.87)	40.98 (23.37)
LFSW	50.32 (18.43) <sup>b*</sup>	30.58 (22.21) <sup>b*</sup>	49.00 (23.09)	43.17 (21.19)

\* $p < 0.05$

Note: <sup>a-b</sup> indicate the significant post-hoc comparisons for the interaction between binge type, food type and condition.

#### 7.4.6.3 Implicit wanting

Table 7.5 displays the outcome of the implicit wanting trials. In the fasted condition, O-B responded faster for HFSW and HFSA food items, whereas O-NB displayed relatively low implicit wanting for all food categories compared to O-B in both conditions. Interestingly, implicit wanting for HFSW was lower in O-NB in the fed condition compared to the fasted condition, whereas it was higher in O-B. When

fasted, L-B responded faster for the LFSW and LFSA food categories. In comparison to this L-NB, responded faster for both savoury categories when fasted. In the fed condition, implicit wanting for the HFSW and the LFSW food items were greater in both lean types compared to the fasted condition.

There was a main effect of food type [ $F(3, 126) = 3.03, p < 0.03$ ] in which the LFSW category appeared to have the greatest implicit wanting, however this finding did not withstand more stringent post hoc analysis. Additionally, there was an interaction between condition and food type [ $F(3, 126) = 4.78, p < 0.01$ ]. Post hoc analyses revealed that in the fasted condition, participants responded faster for LFSA food items and in the fed condition they responded faster for the two sweet food categories. Finally, there was an interaction between binge type and food type [ $F(9, 126) = 2.28, p < 0.02$ ] with greater implicit wanting for HFSW in O-B, for LFSW in L-B, whereas the opposite pattern was apparent in the O-NB who had greater implicit wanting for LFSA and in L-NB who had greater implicit wanting for HFSW.

Table 7.5 Mean (standard deviation) implicit wanting (D-RT) for O-B, O-NB, L-B and L-NB for the food categories in the fasted and fed condition

Fasted	O-B (n=11)	O-NB (n=12)	L-B (n=12)	L-NB (n=11)
HFSA	.046 (.369)	.058 (.462)	-.240 (.759)	.210 (.478)
LFSA	-.156 (.411)	.023 (.417)	.126 (.397)	.146 (.394)
HFSW	.125 (.180)	.048 (.582)	-.113 (.357)	-.114 (.526)
LFSW	-.015 (.478)	-.043 (.503)	.225 (.474)	-.245 (.677)
Fed	O-B (n=11)	O-NB (n=12)	L-B (n=12)	L-NB (n=11)
HFSA	-.254 (.582)	.004 (.409)	-.639 (.509)	-.133 (.447)
LFSA	-.279 (.574)	.049 (.359)	.115 (.479)	-.245 (.482)
HFSW	.285 (.479)	-.140 (.573)	.242 (.349)	.139 (.512)
LFSW	.248 (.324)	.088 (.529)	.475 (.244)	.131 (.458)

## 7.4.7 Relationship between LFPQ measures and energy intake

### 7.4.7.1 Explicit liking

Table 7.6 shows explicit liking for HFSW was positively associated with overall energy intake and energy intake from sweet foods in the fasted and the fed condition. Explicit liking for LFSW was associated with sweet food intake in the fasted condition and overall and sweet food intake in the fed condition. In the fed condition, explicit liking for HFSA was positively associated with overall energy intake and intake of savoury foods.

Table 7.6 Pearson's correlations between energy intake (kcal) and explicit liking in the fasted and fed condition

	Fasted				Fed			
	HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Overall energy intake	.069	-.193	.396**	.229	.393**	.244	.498***	.325*
Sweet energy intake	-.048	-.076	.390**	.291*	.247	.208	.543***	.390**
Savoury energy intake	.252	-.278	.093	-.079	.456***	.091	.085	.023

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

### 7.4.7.2 Explicit wanting

As shown in Table 7.7, explicit wanting for HFSW was positively associated to overall energy intake and intake from sweet foods in both conditions. Explicit wanting for LFSW was positively associated with overall energy intake and sweet intake in the both conditions. In the fed condition, explicit wanting for HFSA was positively associated with overall energy intake and intake from savoury foods.

Table 7.7 Pearson's correlations between energy intake (kcal) and explicit wanting in the fasted and fed condition

	Fasted				Fed			
	HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Overall energy intake	.104	-.202	.421**	.306*	.403**	-.252	.472***	.301*
Sweet energy intake	-.059	-.141	.382**	.347*	.219	-.189	.485***	.349*
Savoury energy intake	.355*	-.165	.166	-.020	.485***	-.146	.170	.044

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

#### 7.4.7.3 Implicit wanting

Implicit wanting for HFSW was positively associated with sweet food intake in the fasted and fed condition, whereas in the fed condition the association between implicit wanting for HFSW and overall energy intake approached significance [ $r(46) = .281, p = 0.06$ ]. Implicit wanting for LFSW was associated with overall energy intake and energy intake from sweet foods (see Table 7.8).

Table 7.8 Pearson's correlations between energy intake (kcal) and implicit wanting in the fasted and fed condition

	Fasted				Fed			
	HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Overall energy intake	.032	-.273 <sup>1</sup>	.256	-.038	.035	-.128	.281 <sup>1</sup>	.357*
Sweet energy intake	-.112	-.199	.298*	.013	-.082	-.126	.300*	.370*
Savoury energy intake	.302*	-.208	-.033	-.111	.269	-.051	.034	-.216

\* $p < 0.05$ ; <sup>1</sup> $p = 0.06$

## 7.5 Discussion

The present study examined food reward and food intake during fasted and fed states according to individual differences in body mass index (overweight or obese and

lean) and trait binge eating (binge-type or non-binge type). It was hypothesised that lean and obese binge types would be characterised by enhanced implicit wanting for high-fat sweet foods, and enhanced liking for food overall compared to the lean and obese non-binge types. Additionally, it was hypothesised that trait binge eating would be associated with increased intake of sweet foods in the ad libitum eating task.

The manipulation of motivational state was successful with all participants reporting greater levels of hunger in the fasted condition compared with the fed condition. In line with this, participants consumed more energy when fasted than when fed. Consistent with previous research (Epstein, Temple, et al., 2007b; Nijs et al., 2010; Saelens & Epstein, 1996), it was shown that age-matched overweight and obese women consumed more energy ad libitum compared to lean controls. Furthermore, intake was greater independent of condition. It is important to note that these differences in intake were not due to increased levels of hunger experienced by the overweight and obese individuals, as there were no differences in self-reported appetite sensations. Indeed, a strength of the current study was that the test meal was individually calibrated to provide each participant with 25% of their daily energy requirements, accounting for differences in estimated energy needs of overweight or obese and lean individuals.

When the influence of trait binge eating was examined, only the obese-binge types (O-B) were shown to consume more energy compared to the other groups. O-B's increase in energy intake was mostly accounted for by a greater consumption of sweet foods. A similar preference for sweet foods was also evident in the lean-binge type (L-B), although this was not associated with greater overall energy intake, which is consistent with the findings from Chapter 6. This increase in overall energy intake in O-B was not due to increased levels of hunger as there was no effect of trait binge eating on ratings of appetite sensations or on the satiating efficiency of food.

In addition to increased intake of sweet foods in the ad libitum eating task, O-B reported experiencing a greater degree of food craving intensity, specifically for sweet foods over the previous seven days when compared with O-NB. This finding is consistent with previous reports (Greeno et al., 2000) and perhaps suggests that overweight or obese individuals with the tendency to binge eat may have a greater preference or motivation for sweet foods outside of the laboratory which may lead to increased consumption of sweet foods in their habitual diet. Furthermore, O-B scored lower on the Positive Mood subscale of the COEQ. Contrary to expectations, there were no differences between L-B and L-NB in self-reported food cravings.

Interestingly, O-B had a larger waist circumference compared to O-NB, with no corresponding difference in body mass index. A large case-control study on risk factors for myocardial infarction found that measures of waist-to-hip ratio and waist circumference were better predictors of myocardial infarction than BMI (Yusuf et al., 2005). Therefore, the increase in waist circumference in O-B may enhance their risk of obesity-related health problems which central adiposity has been shown to predict (Zhu et al., 2002). These findings highlight the importance of examining markers of adiposity in addition to BMI when defining obesity (and relevant subtypes).

In addition to examining food intake and food choice, the role of underlying reward processes were examined by comparing measures of explicit liking, explicit wanting and implicit wanting in lean and overweight or obese binge and non-binge types. It was shown that, when comparing the overweight or obese groups, O-B had an enhanced liking for all foods compared to O-NB, which appeared to be independent of their motivational state. Research investigating a link between enhanced liking for foods and susceptibility to weight gain and overeating has been inconsistent to date. Blundell et al., (2005) found that individuals who were overweight or obese, and habitually consumed a high fat diet, rated food as more pleasant compared to those consuming a similar diet but were lean, while other research has reported no

differences between overweight or obese and lean individuals' liking for food (Cox et al., 1999; Saelens & Epstein, 1996). However, the tendency to binge eat has been shown to be associated with enhanced liking in both obese individuals with BED (Davis et al., 2009) and lean individuals with moderate levels of binge eating severity (Finlayson et al., 2011).

The findings for the implicit wanting trials were mostly consistent with the outcome of Chapter 6. Enhanced implicit wanting for high-fat sweet foods was evident in L-B when compared to L-NB in the fed condition. However, unlike in Chapter 6, L-B had greater implicit wanting for low-fat sweet foods rather than high fat sweet foods in the fasted condition suggesting that although the preference for sweet taste was still present, the preference for the combination of fat and sweet was not. This raises the question as to whether L-B is characterised by an enhanced implicit wanting for high fat sweet foods specifically, or sweet taste in general.

Consistent with the hypothesis, O-B had greater implicit wanting for high-fat sweet foods compared to O-NB. Interestingly, O-B responded faster for the high-fat sweet category compared to the other categories in both the fasted and fed conditions. This increase in implicit wanting for high-fat sweet foods in the fed condition was also seen in L-B and L-NB but it was only the O-B that exhibited an enhanced motivation exclusively for sweet foods in both conditions supporting the notion that trait binge eating is characterised by an increased liking and wanting for sweet foods that is independent of motivational state. Coupled with the increased self-reports of food craving, it can be suggested that this increased hedonic response for high-fat sweet foods seen in the O-B may convey a risk of further weight gain. This suggestion is supported by recent evidence, which demonstrated that trait binge eating was positively associated with increases in fat mass over a period of one year in a sample of first year undergraduate students (Finlayson, Cecil, et al., 2012).

Interestingly, O-NB's implicit wanting scores indicated that they did not have an enhanced wanting for any of the food categories in either condition. Similarly, in



both conditions their ratings of explicit liking were very similar for all of the food categories. While this finding was unexpected, it may be that these individuals were unique in that they did not have a strong preference for any particular *type* of food assessed in the current study. However, it should be noted that they did not seem to dislike any of the foods, as their ratings of explicit liking were not unusually low compared to the other groups. These findings raise further questions about the sensitivity of studies on eating behaviour that compare lean and obese groups based on BMI classification alone. Obesity is a highly heterogeneous condition and phenotypes based on potent biopsychological traits are likely to interact with food quite distinctly.

When the association between food hedonics and energy intake were assessed it was revealed, consistent with the findings of Chapter 5, that energy intake of sweet foods was positively related to ratings of liking and wanting for these types of foods. Further to this, the relationship between explicit ratings and energy intake were considerably stronger in the fed compared to the fasted condition. Notably, greater explicit ratings of liking and wanting for high-fat savoury foods were associated with greater intake of these foods in the fed condition only. This finding is consistent with the notion that an increased hedonic response to palatable foods may lead to increased intake even when hunger is suppressed.

Although the present study carried a number of strengths, there were also some limitations to consider. First, the sample consisted of females recruited from or near a University campus therefore the applicability of the findings to the wider population may be limited. However, steps were taken to ensure that those recruited consisted of an equal ratio of student and non-student women. Furthermore, as the study was only conducted in females, it is necessary to conduct further research to examine whether the findings are likely to generalise to males. A strength of this study was that it was conducted in a controlled setting and that there was a wash out period of at least seven days between conditions. However with this increased

control and precision over study variables, the artificial setting may also reduce the applicability of the findings to the free-living environment.

In summary, the current study provides evidence that trait binge eating forms part of a hedonic phenotype susceptible to overconsumption in overweight and obese females. This phenotype appears to be characterised by differences in energy intake, food choice, liking and wanting, and experiences of food craving. While the findings of the current study were not wholly consistent with Chapter 6 with regards to the lean individuals (which may be attributable to differences in study design), the increased preference for sweet food, and increased implicit wanting for sweet foods were present in L-B demonstrating a degree of robustness to these findings. Finally, an interesting question was raised from the findings as to whether the preference observed in the lean groups concerns sweet taste specifically or a combination of fat and sweet, which appeared to be most preferred by the obese binge type.

## **7.6 Summary**

- Overweight or obese individuals consumed more energy in the ad libitum eating task compared to their lean counterparts. However, when the influence of trait binge eating was taken into consideration it was revealed that only O-B consumed more energy.
- O-B displayed greater liking for all foods and greater implicit wanting for high-fat sweet foods in the fasted and fed condition. Furthermore, they consumed more high-fat sweet foods.
- O-B reported experiencing greater craving intensity and craving for sweet foods over the previous seven days compared to O-NB. In addition, they reported experiencing lower levels of positive mood.
- O-B had greater levels of central adiposity (assessed by waist circumference) compared to O-NB.
- In line with the findings from Chapter 6, L-B had a greater preference for sweet foods in the ad libitum eating task and a greater liking and implicit

wanting for these foods compared to L-NB. Unlike Chapter 6, increased implicit wanting for HFSW foods were not present in the fasted condition but implicit wanting for LFSW was. This suggests that in lean individuals trait binge eating may be associated with increased preference for sweet taste rather than a combination of sweet and fat.

## Chapter 8<sup>6</sup>

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### **48-hour assessment of trait binge eating and ‘food addiction’ using combined laboratory and free-living measures of eating behaviour**

#### **8.1 Abstract**

The current study had two major aims: 1) to determine whether the previous findings in overweight or obese individuals with high or low levels of trait binge eating extend beyond the acute laboratory situation and relate to eating behaviour in the natural setting; and 2) to utilise a novel psychometric questionnaire - the Yale Food Addiction Scale (YFAS) - and explore whether its construct of ‘food addiction’ was able to identify a subtype of disordered eating that was distinct from binge eating tendency. Using a matched pairs design, twenty-four overweight or obese females and ten lean females were recruited on the basis of their score on the Binge Eating Scale (BES) to form four distinct groups; Obese ‘binge type’ (O-B); Obese ‘non-binge type’ (O-NB); Lean ‘binge type’ (L-B) and Lean ‘non-binge type’ (L-NB). Energy intake was assessed over 48-hours using combined laboratory-based test meal methodology and free-living dietary recall procedures. O-B and L-B exhibited a greater preference for sweet snack foods in their laboratory and free-living eating behaviour. This was supported by greater laboratory-based measures of wanting and craving for this food type in O-B. A subgroup of O-B met the YFAS criteria for ‘food addiction’. These individuals exhibited the highest BES scores, greater levels of eating related distress, enhanced food cravings, greater energy intake and different food preferences compared to O-B who did not meet the criteria for ‘food addiction’. These findings firstly support the use of trait binge eating as a common hedonic phenotype of obesity and extend the relevance of this phenotype to habitual patterns of energy intake. Secondly, these findings provide evidence that ‘food addiction’, as defined by the YFAS, appears to fit along the continuum of the

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<sup>6</sup> Parts of this chapter are based on a study that has been published “Dalton, M., Blundell, J. & Finlayson, G. (2013) Examination of obese binge-eating subtypes on reward, food choice and energy intake under laboratory and free-living conditions. *Frontiers in Psychology*, 4, 757.”

Binge Eating Scale and may correspond to a more severe expression of binge eating tendency. Therefore the classification of certain individuals as 'food addicts' may not be appropriate.

## **8.2 Introduction**

### **8.2.1 Free-living versus laboratory-based study of eating behaviour**

Findings from laboratory-based studies examining the eating behaviour of individuals with or without Binge Eating Disorder (BED) have consistently shown that compared to obese controls, individuals with BED consume more energy in ad libitum eating tasks (Geliebter et al., 2001; Goldfein et al., 1993; Latner et al., 2009; Yanovski et al., 1992). Assessing eating behaviour in the laboratory has many advantages due to the increased levels of experimental control, precision and accuracy in the measurement of energy and nutrient intake that cannot typically be attained using more naturalistic, ecologically valid procedures. However, there is a trade-off between precision and naturalness, as the laboratory environment tends to constrain participants' eating behaviour (Blundell et al., 2009) and by using laboratory techniques alone we cannot be confident that the eating behaviour observed will generalise to free-living eating behaviour.

Research utilising food diaries and food recall procedures have the advantage of assessing eating behaviour over longer periods of time than typical laboratory studies and allow for the study of more natural eating behaviour. In general, evidence from dietary recall procedures accord with the findings from laboratory studies. For example, Raymond, Neumeyer, Warren, Lee, and Peterson (2003) examined 24-hour dietary recalls of individuals with or without BED and found that on days when a binge occurred, those with BED reported consuming more energy than those without BED, particularly during the evening. Interestingly, however, there were no group differences in self-reported energy intake on days when a binge did not occur. Allison and Timmerman (2007) examined free-living eating behaviour in a sample of binge eating females over 14-days using food diary methodology. They found an

increased incidence of binge eating during lunch and dinner periods and on weekends, with popular binge foods including bread, pasta, sweet foods and high-fat meats. Further to this, Allison and Timmerman reported that BMI was positively associated with binging during meals rather than on snack foods, and that those individuals who predominately binged on sweet foods reported a greater number of binge days than individuals who binged on other types of food.

While self-report measures have the scope to represent free-living eating behaviour there are several limitations that impact the validity of the information gathered. With regards to the use of food diaries, the reliability and validity of the data collected tends to deteriorate as the length of time the record is kept increases due, in part, to the increased burden on respondents (Gersovitz, Madden, & Smiciklas-Wright, 1978). In addition, self-report measures tend to provide an underestimation of energy intake due to under- or misreporting by the subject (Hill & Davies, 2001; Martin et al., 1996) with research suggesting that the rate of underreporting increases with BMI (Moshfegh et al., 2008).

Relatively few studies have attempted to combine laboratory eating behaviour measures with free-living eating behaviour measures in order to assess the extent to which behaviours observed under controlled laboratory conditions can be compared to behaviour in the natural setting. The findings from Chapter 7 demonstrated how variation in trait binge eating in overweight or obese individuals was associated with greater food consumption, especially of high-fat sweet foods in an ad libitum eating task. However it is not known whether trait binge eating is associated with more habitual overconsumption as the increased intake in the laboratory in the obese 'binge-types' may have resulted in a compensatory reduction in energy intake later in the day. Additionally, it was demonstrated that greater trait binge eating scores were related to enhanced implicit wanting for high-fat sweet foods and greater liking for food overall in both fed and fasted states. Therefore, in order to extend these findings, the present study aimed to examine food intake during two 24-hour

periods: firstly using test meal methodology in the laboratory; and secondly using a validated dietary recall technique, in the natural, unrestricted setting.

### **8.2.2 Emergence of food addiction as a subcomponent of obesity**

The debate over whether certain eating disorders, in particular BED, should be redefined as a form of addiction has been present in the scientific literature for over two decades (Cassin & von Ranson, 2007; Davis & Carter, 2009; Davis & Claridge, 1998; Davis, Curtis, et al., 2011; Gearhardt et al., 2011; Haddock & Dill, 2000; Wilson, 1991, 2010). However, it is only in recent years that the notion of ‘food addiction’, as a valid and genuine biopsychological disorder, has emerged as a contentious social, political and scientific issue. This is perhaps due in part to neurobiological evidence that suggests when rats are fed a high sugar diet, they display behaviours that are consistent with several behavioural indicators of drug dependence when the diet is withdrawn (Avena & Hoebel, 2003; Avena et al., 2005; Avena, Rada, & Hoebel, 2008, 2009; Hoebel, Avena, Bocarsly, & Rada, 2009; Hoebel, Rada, Mark, & Pothos, 1999). In addition, the development of the Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009), based on the DSM-IV criteria for substance dependence, has provided the means for the investigation of dependence-like behaviours in humans. The recent validation of the YFAS in individuals with BED (Gearhardt et al., 2011) allows for the possible refinement of the binge eating subtype characterised in the current thesis.

Preliminary evidence using the YFAS in a sample of young normal weight adults has suggested that the scale has high convergent validity with other measures of eating disturbances – including the Binge Eating Scale (Gormally et al., 1982) – and a degree of incremental validity in explaining binge eating tendencies (Gearhardt et al., 2009). Further research by Gearhardt and colleagues has shown that the YFAS appears to distinguish a more severe subtype of BED, that is associated with greater levels of negative affect, lower self-esteem and more frequent binge eating episodes (Gearhardt et al., 2013; Gearhardt et al., 2011). In addition, Gearhardt et al. (2011)

demonstrated that when the YFAS was used as a continuous measure (by considering the symptoms rather than the dichotomous diagnosis of food addiction) it explained 6.3% unique variance in binge eating scores. Davis, Curtis, et al. (2011) corroborated these findings showing that obese individuals who met the YFAS diagnostic criteria for food addiction had greater co-morbidity with BED. Furthermore, obese individuals with food addiction were more impulsive, experienced a greater number of depressive symptoms and reported greater food cravings than those who did not meet the criteria for food addiction (Davis, Curtis, et al., 2011). These findings suggest that behaviours and experiences that are proposed to reflect addictive tendencies towards food, as defined by the YFAS, may distinguish a relevant, possibly distinct, subtype among those who show binge eating tendencies.

### **8.2.3 Study aims**

The current study was designed with two primary aims. The first was to determine whether the previous findings in overweight or obese individuals with high or low levels of trait binge eating extend beyond the laboratory situation and relate to free-living eating behaviour in the participants' natural setting. To do this food intake was examined over two 24-hour periods: one under laboratory conditions using test meal methodology, and one under free-living conditions using a validated 24-hour dietary recall technique. The second aim was to explore the recent idea that 'food addiction', as defined by the YFAS, may form a subtype of disordered eating distinct from binge eating tendency. It was hypothesised that greater levels of binge eating would be associated with greater energy intake and a preference for high-fat sweet foods in the laboratory and in the participants' natural setting. Furthermore, in line with the previous findings in this thesis, it was hypothesised that 'binge-types' would have greater liking for foods and greater implicit wanting for high-fat sweet foods compared to 'non-binge types'.



## **8.3 Method**

### **8.3.1 Participants**

Twenty-four overweight or obese (age:  $25.42 \pm 6.42$ , BMI:  $30.30 \pm 2.60$ ) and ten lean (age:  $24.80 \pm 3.65$ , BMI:  $20.62 \pm 1.66$ ) females were recruited from the staff and student population at the University of Leeds. Participants with a BMI between 18.5 and 24.9 were classified as lean, and participants with a BMI between 27.5 and 35 were classified as overweight or obese. Participants were selected from an initial screening process that firstly excluded those who were taking medication, currently dieting, reported a history of eating disorders, or were unfamiliar with or disliked any of the study foods. Secondly, participants were screened on the basis of their score on the BES. In the overweight or obese group, twelve individuals scoring  $\geq 17$  formed the “obese binge type” group and twelve individuals scoring  $\leq 6$  formed the “obese non-binge type” group. In the lean group, five individuals scoring  $\geq 14$  formed the “lean binge type” group and five individuals scoring  $\leq 6$  formed the “lean non-binge type” group. The groups were individually matched by age, with an overall age difference of no more than three years across each matched pair (see Figure 8.1). Four participants from the study presented in Chapter 7 (2 who were in the O-B group and 2 who were in the O-NB group) completed the screening questionnaire for the current study and were invited to participate. Informed written consent was obtained prior to the study. Participants received £15 for their participation. All research procedures were reviewed and approved by the University of Leeds, Institute of Psychological Sciences.

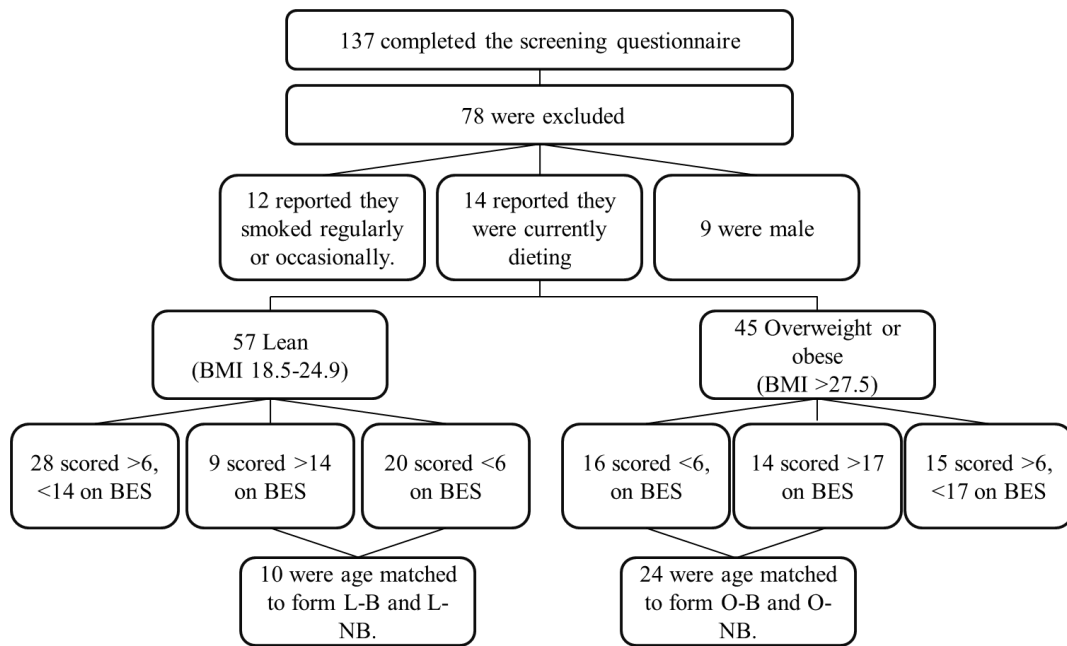


Figure 8.1 Outcome of the recruitment process and reasons for pre-study exclusion

### 8.3.2 Design

A between subjects (high or low binge eating score) design was used with participants attending the research unit on two occasions over the course of three days. The first visit involved a 24-hour period of energy intake measured using test meal methodology in the laboratory (referred to as test meal methodology energy intake [TM-EI] from this point forward). Participants were required to have fasted from 9pm the evening before so that accurate measurements of height, weight, waist circumference and body composition could be taken. During TM-EI, participants came to the research unit for their breakfast, lunch and dinner. Each eating occasion was separated by a period of four hours. During the remaining time spent outside of the HARU participants were required not to eat or drink any food or beverages, except water, unless provided by the researcher. Throughout TM-EI, participants used a validated, hand-held Electronic Appetite Ratings System (EARS II; HP iPAQ) to complete hourly ratings of appetite and mood (Gibbons et al., 2011). The EARS II provided a time stamp for each entry so compliance with this instruction was monitored.

The second visit was held two days after TM-EI. During this visit (referred to as dietary recall energy intake [DR-EI] from this point forward) free-living energy intake was assessed for the previous 24 hours using the validated Automated Multiple Pass Method (Moshfegh et al., 2008). Participants were asked to recall all food and beverage items consumed from the time they left the laboratory on TM-EI to 10pm the evening before DR-EI. The purpose of DR-EI was not disclosed in order to reduce the likelihood of participants intentionally (for the purposes of the study) monitoring, restricting or rehearsing their food intake during the 24-hour dietary recall period. The DR-EI visit was held on either a Wednesday or Thursday to avoid weekend fluctuations in energy intake. The two test sessions were conducted in the follicular phase of the participants' menstrual cycle in order to minimise the influence that the luteal phase may have had on energy intake and food choice (Cohen, Sherwin, & Fleming, 1987; Dye & Blundell, 1997). In addition, participants binge type group status was assigned following the initial screening questionnaire and then stored until after the data were analysed in order to reduce the impact of researcher bias during TM-EI and DR-EI and subsequent data entry (including entry of the 24-hour dietary recall).

### **8.3.3 Measures**

#### **8.3.3.1 Subjective appetite and mood sensations**

Measures of hunger, fullness, desire to eat, prospective consumption, irritability and contentedness were taken using the EARS II. Questions concerning subjective appetite sensations were the same as those described in Chapter 5. To assess mood, participants were asked "How irritable do you feel right now?" and "How content do you feel right now?" both questions were anchored with the statements "not at all" and "extremely".

#### **8.3.3.2 Food hedonics: explicit liking and implicit wanting**

Measures of explicit liking and implicit wanting were assessed using the LFPQ, which is described in greater detail in Chapter 4.

### **8.3.3.3 Psychometric questionnaires**

#### **8.3.3.3.1 Binge eating scale**

The Binge Eating Scale (BES; Gormally et al., 1982) was completed during initial online screening and was used to assign participants to either the binge type or the non-binge type groups.

#### **8.3.3.3.2 Three factor eating questionnaire**

The Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) was used to assess levels of restraint, disinhibition and hunger and was completed at the end of the study procedures.

#### **8.3.3.3.3 Control of eating questionnaire**

The Control of Eating Questionnaire (COEQ; Hill et al., 1991) was completed at the end of the study procedures in order to assess participants' level of positive mood and experience of food cravings over the previous seven days.

#### **8.3.3.3.4 Yale Food Addiction Scale**

The Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009) was given to participants at the end of the study procedures in order to assess behaviours and experiences associated with addictive eating behaviour.

### **8.3.3.4 Energy intake measures**

#### **8.3.3.4.1 Ad libitum test meal intake**

Participants consumed breakfast, lunch and dinner in the laboratory using test meal methodology. All foods were provided in ad libitum quantities (see Table 8.1) and participants were provided with plates and bowls in order to allow them to serve themselves. Prior to each meal, participants were instructed to eat until they were comfortably full. Participants ate alone in an experimental cubicle with water provided ad libitum. For the lunch test meal, participants were provided with two types of sandwich and each sandwich was sliced into quarters. To aid with the

calculation of energy intake, participants were informed, before they began eating, that they should finish any quarters they began to eat. Food was measured to the nearest 0.1 g and energy values were determined using food tables and manufacturer labelling.

Table 8.1 Food items and quantities provided for breakfast, lunch and dinner on the laboratory energy intake day.

Breakfast		Lunch		Dinner	
Cornflakes*	175g	Cheese sandwich		Pasta	300g
Branflakes*	175g	Grated cheese	45g	Pasta sauce	475g
Milk	500ml	Margarine	10g	Grated cheese	100g
Wholemeal bread*	184g	Bread	92g	Plain baguette	85g
White bread*	184g	Cream cheese sandwich		Garlic baguette	85g
Flora spread	30g	Low-fat cream cheese	34g	Lettuce	150g
Strawberry jam	30g	Margarine	10g	Tomatoes	115g
Granulated sugar	50g	Bread	92g	Cucumber	115g
		Strawberry yoghurt	300g	Chocolate rolls	80g
		Cheese savouries	100g		

*Note:* \*Participants selected either Cornflakes or Branflakes, and either white or wholemeal bread. Tea and coffee were provided upon request with each meal with optional milk and sugar.

#### 8.3.3.4.2 Ad libitum snack intake

To provide a measure of ad libitum snack food intake participants were given a “snack box” which contained four pre-selected snack foods. The snacks foods represented the categories of food items presented in the LFPQ and participants selected one item from a choice of three from each category (see Table 8.2) during screening. To do this, the participants first ranked each snack food from “most preferred” to “least preferred” and then rated each item for pleasantness and frequency of consumption using seven-point Likert scales. Participants received 100g of each item in clear plastic food bags, which were placed in a jute bag for them to take away and consume if and as they wished, in the four-hour period between lunch and dinner. Participants were told that they could consume as much or as little as they wanted from the bag, but that they should not share, give away or dispose of the items. The snack box was collected at the beginning of the dinner

session. Food from the snack box was measured to the nearest 0.1 g and energy values were determined using manufacturer labelling.

Table 8.2 Food items available for selection in the ad libitum snack box.

High-fat savoury	Low-fat savoury	High-fat sweet	Low-fat sweet
Mini cheddars	Snack-a-jacks	Chocolate buttons	Jelly babies
Crisps	Salted pretzels	Mini cookies	Wine gums
TUC crackers	Ryvita thins	Flapjack	Fruit pastilles

#### **8.3.3.5 24 hour dietary recall**

The United States Department of Agriculture's Automated Multiple Pass Method (AMPM; Moshfegh et al., 2008) was used as a measure of the participants' free-living main meal and snack food intake over the second 24-hour period and was administered during DR-EI. The AMPM consists of five stages, which are described in Chapter 4. Measuring cups, spoons and images of food portions were provided to aid with the estimation of portion size.

#### **8.3.3.6 Estimation of daily energy expenditure**

Estimated daily energy expenditure was calculated using the Schofield equations (Schofield, 1985) for basal metabolic rate multiplied by physical activity level (PAL) from self-reported frequency and mode of exercise performed per week. Estimated daily energy expenditure was used to assess whether participants had consumed more than their daily energy requirements.

#### **8.3.3.7 Body composition**

Air plethysmography (Bodpod, Concord, CA, USA) was used in order to obtain an estimate of participants' fat mass, fat free mass and percentage body fat. Measures of body composition were taken following an overnight fast with participants wearing non-underwired swimwear and a swim cap.

#### **8.3.4 Procedure**

The participants attended the research unit for two sessions: a test meal methodology energy intake day (TM-EI) and a dietary recall day (DR-EI) (see Figure 8.2). For the TM-EI, participants arrived between 7:00-9:00am after having fasted from 9pm the evening before. Firstly, measurements of height, weight, waist circumference and body composition were taken. The participants then completed baseline appetite and mood ratings after which breakfast was consumed. Following breakfast, the second of the appetite and mood ratings were completed and participants were allowed to leave the research unit. During this time the EARS II prompted completion of hourly ratings of appetite and mood until they were due to return for lunch, the same occurred in the period between lunch and dinner. During the lunchtime session participants completed the LFPQ twice, once before the lunchtime test meal and again ten minutes after they had finished their lunch. Appetite and mood ratings were taken at the start and after each event in the lunchtime procedure. At the end of lunch participants were given the snack box and were instructed to continue completing appetite and mood ratings. At the start of the dinner time session, participants returned the snack box. Final ratings of appetite and mood were taken before and after the dinner test meal. For the DR-EI, participants arrived at the research unit at a time convenient for them on the second day following TM-EI. Participants were told they would be required to recall all of the food and beverage items they consumed from leaving the unit on Day 1 (TM-EI) to 10pm on the evening of Day 2 using the AMPM. After this was completed, the participants filled in the TFEQ, the YFAS and the COEQ. They received written and verbal debriefing and were compensated for their time before leaving the study.

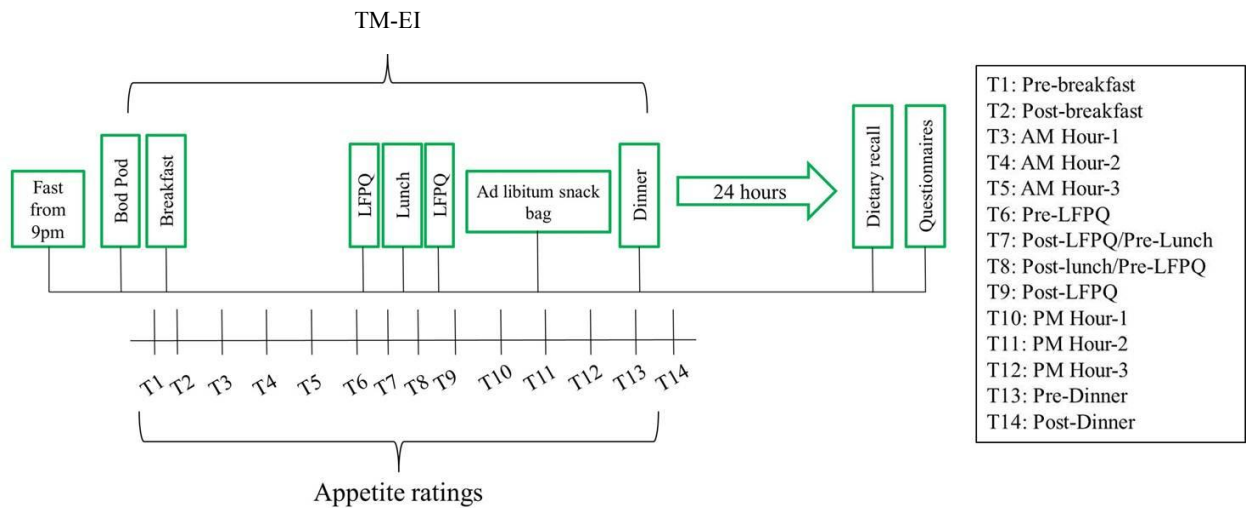


Figure 8.2 Study design

### 8.3.5 Data Analysis

Data were analysed using SPSS version 20 for Windows and are presented as means with standard deviations. To examine the influence of trait binge eating on appetite, mood and the satiety quotient ratings repeated measures ANCOVA were conducted with BES used as a covariate. Independent t-tests were used to examine the influence of trait binge eating within weight groups (i.e. O-B versus O-NB; L-B versus L-NB). The effect of trait binge eating on energy intake from the snack foods during TM-EI was analysed for overall energy intake, energy intake according to the taste of the snack foods (sweet or savoury), and energy intake according to the taste and fat content (high or low) of the snack foods using a 2x2 and a 2x2x2 repeated measures ANOVA, respectively. The number of sweet and savoury processed (not fruit or vegetables) snack foods consumed during DR-EI that were not consumed as part of breakfast, lunch or dinner, were analysed using independent t-tests. Food hedonics were analysed according to motivational state (fasted or fed), food type and binge-type group using two 2x4 ANOVAs for the overweight or obese and the lean groups. Pearson's correlations were used to examine the relationship between laboratory-based and free-living measures of energy intake.



## 8.4 Results I

### 8.4.1 Sample characteristics

Group characteristics of age, anthropometrics, body composition and psychometric traits are shown in Table 8.3. As expected trait binge-eating score was greater in O-B compared to O-NB [ $t(22) = 17.39, p < 0.001$ ], and in L-B compared to L-NB [ $t(8) = 16.02, p < 0.001$ ]. In addition, O-B had higher TFEQ disinhibition [ $t(22) = 4.03, p < 0.001$ ] and hunger [ $t(22) = 3.21, p < 0.01$ ] scores compared to O-NB. While there were no differences in BMI, O-B had greater fat mass than O-NB [ $t(22) = 2.21, p < 0.05$ ] and there was a trend towards a difference in waist circumference [ $t(22) = 1.79, p = 0.08$ ].

Table 8.3 Mean (standard deviation) age, anthropometric, body composition and psychometric trait characteristics of the O-B, O-NB, L-B and L-NB

	O-B (n=12)	O-NB (n=12)	L-B (n=5)	L-NB (n=5)
Age	25.67 (7.28)	25.17 (5.75)	25.40 (4.77)	24.20 (2.49)
Height (cm)	169.29 (5.38)	164.98 (5.02)	164.44 (5.27)	167.58 (1.96)
Weight (kg)	90.18 (13.77)*	79.29 (5.92)*	56.52 (4.27)	56.68 (5.35)
BMI (kg/m <sup>2</sup> )	31.48 (4.65)	30.12 (1.55)	21.00 (1.69)	20.24 (1.73)
Fat mass (kg)	36.29 (13.20)*	27.36 (4.73)*	13.90 (1.70)	14.26 (3.48)
Body fat (%)	39.28 (8.63)	34.93 (5.62)	24.62 (2.54)	24.88 (4.38)
Fat free mass (kg)	53.88 (4.56)	50.16 (5.18)	42.58 (3.49)	42.42 (2.50)
Waist (cm)	98.07 (13.56)	90.58 (5.09)	70.77 (1.67)	69.44 (2.43)
Restraint	11.00 (4.35)	7.83 (3.81)	12.40 (4.51)	9.60 (5.94)
Disinhibition	12.08 (3.32)***	7.17 (2.62)***	10.40 (2.30)	6.60 (4.22)
Hunger	9.25 (3.55)**	5.17 (2.62)**	8.00 (4.64)	4.80 (2.78)
Binge eating score	21.08 (2.97) <sup>a</sup> ***	5.00 (1.21) <sup>a</sup> ***	15.80 (1.48) <sup>b</sup> ***	2.40 (1.14) <sup>b</sup> ***

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

*Note:* Comparisons were made within weight groups

### 8.4.2 Subjective appetite sensations

Subjective sensations of hunger, fullness, prospective consumption and desire to eat were measured at fourteen time points during TM-EI (see Figure 8.2) and were analysed to check compliance with the study procedures. There was a main effect of

time on hunger [ $F(13, 390) = 83.40, p < 0.001$ ] with notable significant decreases in hunger ratings following the ad libitum meals of breakfast [ $p < 0.001$ ], lunch [ $p < 0.001$ ] and dinner [ $p < 0.001$ ]. Similar findings were observed for measures of prospective consumption [ $F(13, 390) = 71.08, p < 0.001$ ] and desire to eat [ $F(13, 390) = 64.68, p < 0.001$ ]. Additionally, ratings of hunger increased following the completion of the LFPQ before [ $p < 0.05$ ] and after lunch [ $p < 0.05$ ] and ratings of prospective consumption increased following the completion of the LFPQ after lunch [ $p < 0.01$ ]. There was a main effect of time on ratings of fullness [ $F(13, 390) = 67.35, p < 0.001$ ] with notable significant increases in ratings of fullness following the ad libitum meals of breakfast [ $p < 0.001$ ], lunch [ $p < 0.001$ ] and dinner [ $p < 0.001$ ].

#### **8.4.3 Effect of trait binge eating on subjective ratings of appetite and mood.**

There were no significant differences between O-B and O-NB on ratings of hunger, fullness, prospective consumption or desire to eat during TM-EI [ $F(13, 286) = .554, p > 0.05$ ;  $F(13, 286) = .664, p > 0.05$ ;  $F(13, 286) = .742, p > 0.05$ ;  $F(13, 286) = .770, p > 0.05$ , respectively]. Additionally, there were no significant differences in O-B and O-NB on ratings of irritability [ $F(13, 286) = .237, p > 0.05$ ] or contentedness [ $F(13, 286) = .585, p > 0.05$ ]. However, the between-subjects effect for contentedness approached significance [ $F(1, 22) = 3.19, p = 0.08$ ], which suggested that there was a trend for O-B to report feeling less content compared to O-NB. The lean comparisons yielded similar findings as there were no differences between L-B and L-NB on ratings of hunger, fullness, prospective consumption, desire to eat, irritability or contentedness during TM-EI [ $F(13, 104) = .515, p > 0.05$ ;  $F(13, 104) = .752, p > 0.05$ ;  $F(13, 104) = 1.27, p > 0.05$ ;  $F(13, 104) = 1.40, p > 0.05$ ;  $F(13, 104) = 1.68, p > 0.05$ ;  $F(13, 104) = 1.47, p > 0.05$ , respectively].

##### **8.4.3.1 Satiety Quotient**

There was no effect of BES on the satiating efficiency of the ad libitum breakfast test meal over the course of the morning [ $F(3, 96) = .507, p > 0.05$ ] or on the satiating

efficiency of the ad libitum lunch [ $F(1, 32) = .017, p > 0.05$ ] or dinner test meal [ $F(1, 32) = 2.80, p > 0.05$ ].

#### 8.4.4 Effect of trait binge eating on energy intake and food choice

##### 8.4.4.1 Test meal methodology energy intake day: Ad libitum test meals

Energy intake from the breakfast, lunch and dinner test meals for O-B and O-NB are shown in Figure 8.3 and for L-B and L-NB are shown in Figure 8.4. O-B consumed significantly more calories from the ad libitum dinner compared to O-NB [ $t(22) = 2.19, p < 0.05$ ]. There were no differences in energy intake between the two lean groups.

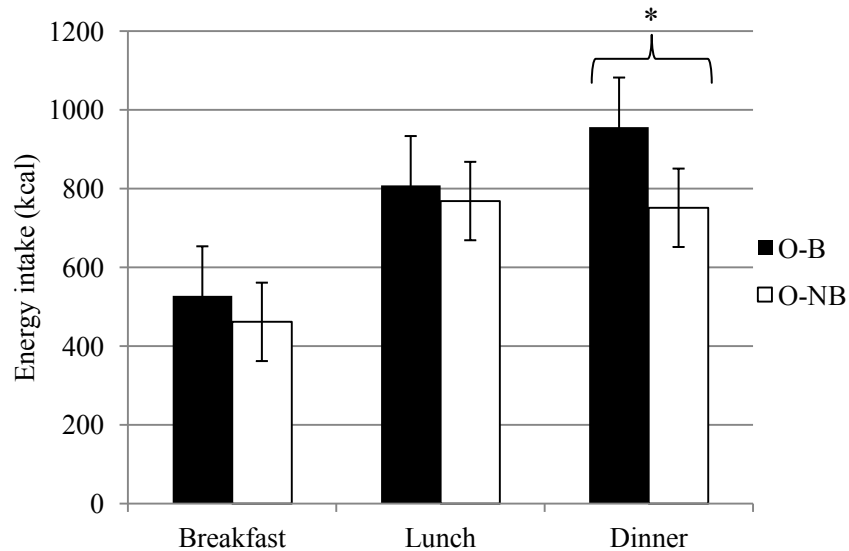


Figure 8.3 Energy intake (kcal) from the ad libitum breakfast, lunch and dinner for O-B and O-NB.

\* $p < 0.05$

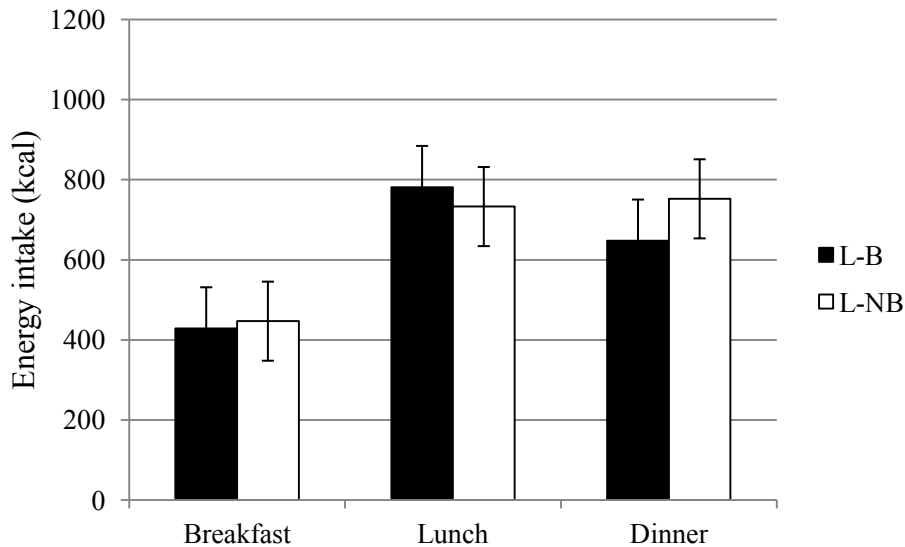


Figure 8.4 Energy intake (kcal) from the ad libitum breakfast, lunch and dinner for L-B and L-NB.

#### 8.4.4.2 Test meal methodology energy intake day: Snack box

The effect of trait binge eating on energy intake from the snack box was analysed for overall energy intake, energy intake according to the taste of the snack foods (sweet or savoury) and energy intake according to the taste and fat content (high or low) of the snack foods.

In the overweight or obese sample, O-B consumed more energy overall from the snack box compared to O-NB [ $F(1, 22) = 6.92, p < 0.02$ ]. There was a main effect of fat [ $F(1, 22) = 18.83, p < 0.001$ ] and taste [ $F(1, 22) = 29.59, p < 0.001$ ] with participants consuming more energy from high fat foods than low fat foods and from sweet foods compared to savoury foods. An interaction between taste and binge type [ $F(1, 22) = 14.43, p < 0.001$ ] revealed that O-B consumed a greater number of calories from sweet foods than O-NB with both groups consuming a similar number of calories from savoury foods (see Figure 8.5). An interaction between fat and binge type [ $F(1, 22) = 4.37, p < 0.05$ ] revealed that O-B consumed more energy from high fat foods (see Figure 8.6) although it appeared that this was accounted for by the increased intake of high-fat sweet foods as this was qualified by an interaction

between fat, taste and binge type [ $F(1, 22) = 4.25, p < 0.05$ ] which showed that O-B consumed 63.4% more calories from high-fat sweet foods compared to O-NB.

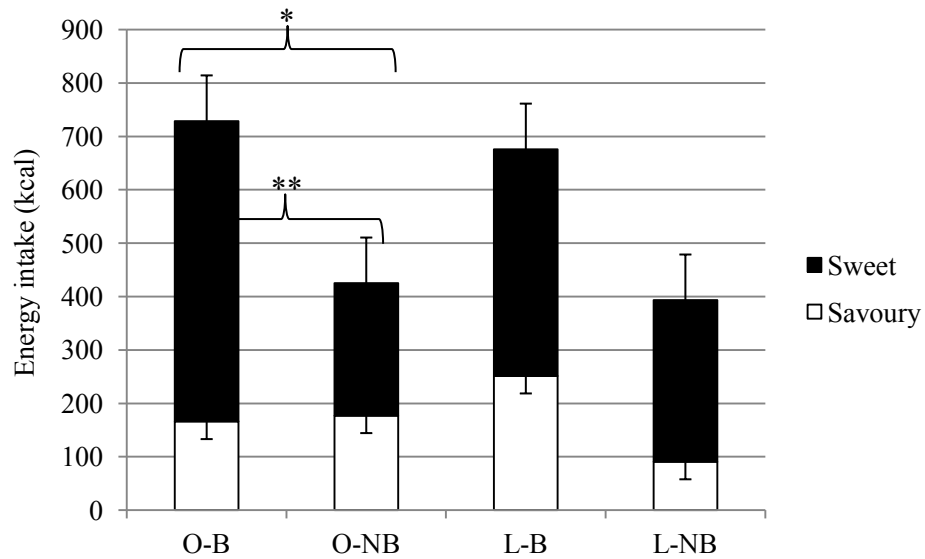


Figure 8.5 Energy intake (kcal) from savoury and sweet snack foods from the ad libitum snack box for O-B, O-NB, L-B and L-NB.

\* $p < 0.05$ ; \*\* $p < 0.01$

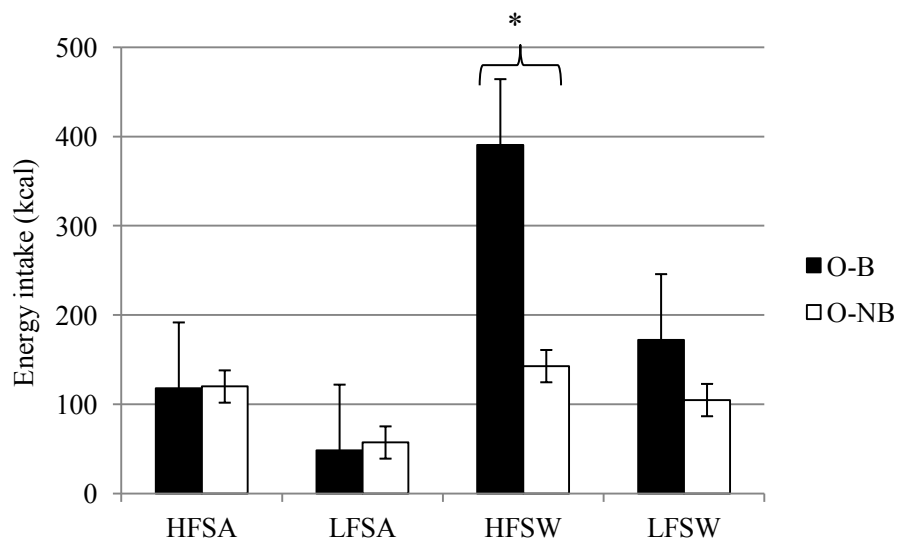


Figure 8.6 Energy intake (kcal) according to fat content and taste of the snack foods for O-B and O-NB.

\* $p < 0.05$

Figure 8.7 shows energy intake according to the fat content and the taste of the food for the lean groups. There was a main effect of taste [ $F(1, 8) = 17.82, p < 0.01$ ] with all participants consuming more energy from sweet foods compared to savoury foods. Additionally, there was an interaction between fat and taste [ $F(1, 8) = 9.58, p < 0.02$ ], which revealed that both L-B and L-NB consumed more energy from high-fat sweet foods.

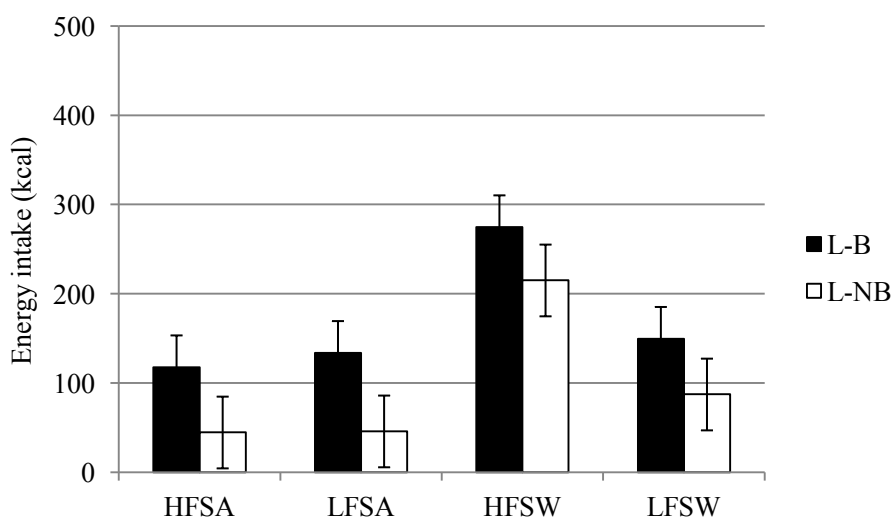


Figure 8.7 Energy intake (kcal) according to fat content and taste of the snack foods for L-B and the L-NB

#### 8.4.4.3 Test meal methodology energy intake day: Evening recall

During DR-EI, participants self-reported what they had consumed during the evening of TM-EI, up to 10pm following the dinner test meal. This was analysed in order to capture any additional intake within the fixed, 24-hour period. Four participants from O-NB, three participants from O-B, three participants from L-NB and two from L-B reported eating nothing during the evening and were excluded from this analysis. O-B ( $528.89 \pm 307.58$ ) reported consuming a greater amount of energy in the evening than O-NB ( $276.48 \pm 201.53$ ) and the difference in energy intake approached significance [ $t(15) = 1.97, p = 0.06$ ]. For the lean groups, L-B

(353.90 ± 162.92) reported consuming more energy during the evening than the L-NB (128.96 ± 112.19) but the difference was non-significant [t (5) = 2.03, p>0.05].

#### 8.4.4.4 Assessment of overconsumption

During TM-EI, O-B consumed significantly more energy than O-NB [t (22) = 3.44, p<0.01]. In addition, L-B consumed more energy over the measured 24-hour period than L-NB but this difference was non-significant [t (8) = .869, p>0.05]. To assess whether O-B were consuming more energy than their estimated daily requirements their estimated 24-hour energy requirements were compared to their TM-EI 24-hour intake (see Table 8.4). There were no differences between the estimated 24-hour energy requirements of O-B and O-NB [t (22) = 1.45, p>0.05] or L-B and L-NB [t (8) = .096, p>0.05]. O-B consumed significantly more than their estimated 24-hour energy requirements compared to O-NB [t (22) = 2.97, p<0.01]. There were no differences between the two lean groups (see Table 8.4).

Table 8.4 Mean (standard deviation) 24-hour energy intake, estimated 24-hour energy expenditure and the mean difference between these for O-B, O-NB, L-B and L-NB.

	O-B (n=12)	O-NB (n=12)	L-B (n=5)	L-NB (n=5)
24-hour energy intake	3417.47 (665.93)	2590.74 (498.19)	2792.25 (718.45)	2486.39 (454.07)
Estimated 24-hour energy requirements <sup>a</sup>	2547.47 (177.31)	2432.99 (208.16)	1979.20 (96.59)	1985.78 (118.80)
Mean difference <sup>b</sup>	870.00 (699.83)	157.75 (448.55)	777.79 (550.80)	476.39 (383.84)
EI:EEE	1.34	1.06	1.41	1.25

*Abbreviations: EI energy intake; EEE estimated energy expenditure*

<sup>a</sup> Estimated 24-hour energy expenditure was calculated using estimated resting metabolic rate multiplied by PAL score for self-reported physical activity levels.

<sup>b</sup> Mean difference was calculated by subtracting actual 24-hr energy intake from estimated 24-hr energy expenditure.

#### 8.4.4.5 Dietary recall day: Main meals and snacks

Table 8.5 shows the self-reported energy intake from DR-EI and includes all reported food and beverages consumed on Day 2 of the procedure (excluding the

self-reported intake from the evening of the TM-EI shown in 8.4.4.3). There was no significant difference in overall self-reported energy intake between O-B and O-NB [ $t(22) = 1.76, p > 0.05$ ] or L-B and L-NB [ $t(8) = .499, p > 0.05$ ]. O-B appeared to consume more energy at breakfast, lunch and from snack foods during DR-EI. The difference in energy consumed at breakfast approached significance [ $t(22) = 2.03, p = 0.055$ ]. For the lean groups, L-B reported consuming more energy at breakfast, dinner and from snack foods compared to L-NB. However, none of these differences in intake were significant.

Table 8.5 Mean (standard deviation) self-reported energy intake for overall energy consumed, and energy consumed at breakfast, lunch, dinner or from snack foods during the dietary recall day.

	O-B (n=12)	O-NB (n=12)	L-B (n=5)	L-NB (n=5)
Breakfast	526.86 (208.52)	369.80 (168.89)	384.07 (79.45)	273.50 (106.85)
Lunch	667.51 (183.75)	566.77 (252.23)	455.68 (169.34)	608.86 (218.97)
Dinner	874.25 (370.74)	1027.81 (282.77)	929.87 (444.49)	634.48 (141.27)
Snacks	628.92 (393.65)	364.62 (395.13)	443.87 (386.14)	281.01 (201.55)
Overall energy intake	2697.54 (644.12)	2329.0 (330.83)	2050.63 (155.31)	1960.71 (371.99)
EI:EEE	1.05	0.96	1.04	0.99

*Abbreviations: EI energy intake; EEE estimated energy expenditure*

The number of processed<sup>7</sup> sweet and savoury snack items consumed during DR-EI were summed and analysed for group differences. In the overweight or obese groups, O-B consumed a greater number of sweet snack items ( $3.5 \pm 1.93$ ) compared to O-NB ( $1.5 \pm 1.0$ ) [ $t(22) = 3.19, p < 0.01$ ]. There were no differences between O-B ( $0.75 \pm .086$ ) and O-NB ( $0.42 \pm 0.68$ ) in the number of savoury snack items consumed. In the lean groups, L-B consumed more ( $1.6 \pm .89$ ) sweet snack items compared to L-

<sup>7</sup> Processed snacks were defined as snack items that were not fruit or vegetables. Items had to be reported as not being consumed as part of breakfast, lunch or dinner.



NB ( $0.80 \pm 1.09$ ) however this difference was non-significant [ $t(8) = 1.27$ ,  $p > 0.05$ ]. There were no differences in the number of savoury snack items consumed [ $t(8) = 1.00$ ,  $p > 0.05$ ] between L-B and L-NB.

#### 8.4.4.6 Dietary recall day: Macronutrient intake<sup>8</sup>

As can be seen in Table 8.6, O-B consumed a greater amount of fat [ $t(22) = 2.26$ ,  $p < 0.05$ ] and saturated fat [ $t(22) = 2.08$ ,  $p < 0.05$ ] compared to O-NB. There were no other group differences and there were no differences between the lean groups.

Table 8.6 Mean (standard deviation) macronutrient intake (grams) for O-B, O-NB, L-B and L-NB from the dietary recall day

	O-B (n=12)	O-NB (n=12)	L-B (n=5)	L-NB (n=5)
Protein (g)	83.23 (19.52)	81.46 (12.62)	82.18 (4.18)	72.49 (17.69)
Carbohydrate (g)	177.65 (56.59)	193.12 (52.26)	121.78 (31.79)	124.68 (20.76)
Sugars (g)	126.64 (52.64)	106.44 (62.96)	113.18 (41.09)	105.68 (15.26)
Fat (g)	69.49 (20.75) <sup>a*</sup>	54.06 (11.41) <sup>a*</sup>	50.81 (11.78)	45.69 (16.23)
Saturated fat (g)	47.21 (17.96) <sup>b*</sup>	34.41 (11.51) <sup>b*</sup>	28.52 (9.91)	28.62 (8.90)

\* $p < 0.05$

#### 8.4.4.7 Effect of trait binge eating on cravings for food

The O-B scored significantly higher on the Craving Intensity [ $t(20) = 2.35$ ,  $p < 0.05$ ] and Craving for Sweet Food [ $t(20) = 2.86$ ,  $p < 0.01$ ] subscales of the COEQ, and lower on the Positive Mood subscale [ $t(21) = 2.26$ ,  $p < 0.05$ ] compared to O-NB (see Figure 8.8).

<sup>8</sup> Macronutrient intake was only analysed for the dietary recall day as in the LEI macronutrient choice could only vary a little due to the limited number of foods offered.

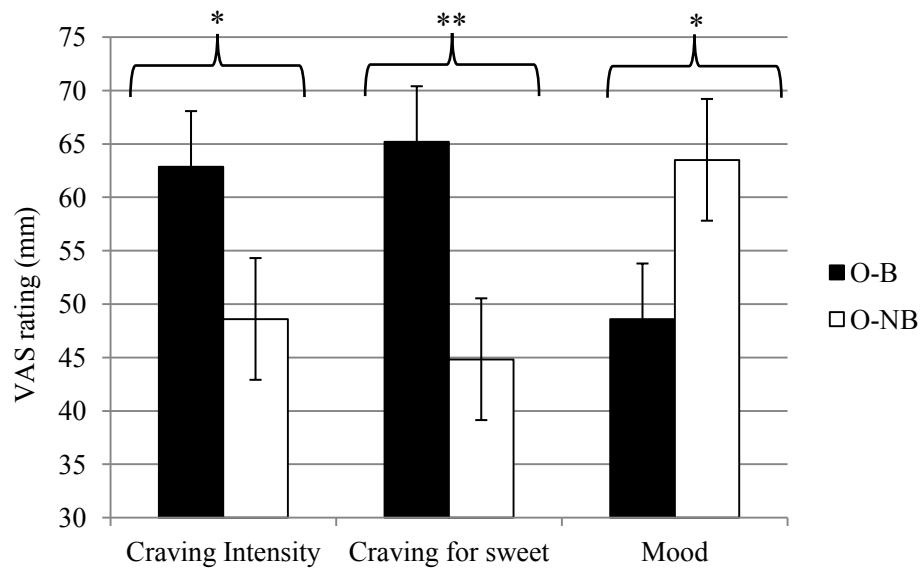


Figure 8.8 Ratings of craving intensity, craving for sweet foods and positive mood over the previous seven days for O-B and O-NB

\* $p < 0.05$ ; \*\* $p < 0.01$

#### 8.4.5 Effect of trait binge eating on food hedonics

##### 8.4.5.1 Explicit liking

Ratings of explicit liking for food were higher for the overweight or obese groups [ $F(1, 22) = 46.72, p < 0.001$ ] and the lean groups [ $F(1, 8) = 10.06, p < 0.01$ ] in the fasted compared to the fed state (see Table 8.7). In the overweight or obese groups there was a food type by binge type interaction [ $F(3, 66) = 3.44, p < 0.02$ ] with O-B having the greatest explicit liking ratings for high fat foods, especially high fat sweet foods when fasted [ $p < 0.03$ ].

Table 8.7 Mean (standard deviation) explicit liking ratings (mm) for O-B, O-NB, L-B and L-NB for the food categories in the fasted and fed state

	O-B (n=12)	O-NB (n=12)	L-B (n=5)	L-NB (n=5)
Fasted				
HFSA	66.67 (15.76)	52.96 (22.95)	55.25 (19.35)	52.00 (38.39)
LFSA	53.94 (22.95)	58.02 (13.74)	56.25 (25.12)	53.65 (23.73)
HFSW	74.52 (15.92) <sup>a*</sup>	50.87 (21.04) <sup>a*</sup>	63.95 (34.12)	44.90 (25.47)
LFSW	64.23 (11.43)	55.89(12.26)	58.95 (18.62)	50.05 (15.22)
Fed				
HFSA	34.44 (22.23)	25.77 (19.88)	29.80 (19.97)	23.30 (16.87)
LFSA	33.83 (20.59)	33.95 (11.21)	22.40 (16.95)	21.00 (19.79)
HFSW	47.39 (21.62)	37.25 (13.09)	59.15 (32.12)	56.10 (27.39)
LFSW	45.04 (15.65)	50.21 (14.08)	57.45 (13.69)	53.10 (31.92)

\* $p < 0.05$

#### 8.4.5.2 Implicit wanting

Table 8.8 displays the outcome of the implicit wanting trials for O-B and O-NB. For both groups implicit wanting for food was greater in a fasted state compared to a fed state [ $F(1, 22) = 15.94, p < 0.001$ ] however a condition by binge type interaction revealed that O-B had enhanced implicit wanting in the fed condition compared to O-NB [ $F(1, 22) = 13.25, p < 0.001$ ]. There was an interaction between food type and binge type [ $F(3, 66) = 5.11, p < 0.01$ ]. Post hoc analyses revealed that O-B responded faster for HFSW foods in both the fasted [ $p < 0.001$ ] and the fed condition [ $p < 0.03$ ]. Finally, there was a three-way interaction between condition, food type and binge type, which approached significance [ $F(3, 66) = 2.45, p = 0.07$ ]. When this was explored, it appeared that O-B responded faster for high-fat sweet foods when fasted compared to O-NB, who responded faster for high-fat savoury food items [ $p = 0.08$ ]. In addition, O-B appeared to avoid low-fat savoury items in both the fasted and fed state condition however only the latter was significant in post hoc analyses [ $p < 0.01$ ].

Table 8.8 Mean (standard deviation) implicit wanting (D-RT) for the O-B and the O-NB for the food categories in the fasted and fed state

	Fasted		Fed	
	O-B (n=12)	O-NB (n=12)	O-B (n=12)	O-NB (n=12)
HFSA	.138 (.688)	.700 (.815)	-.775 (.815)	-.792 (.689)
LFSA	-.636 (.493)	.120 (.585)	-.121 (.752)	-.067 (.867)
HFSW	.228 (.405) <sup>a***</sup>	-.475 (.438) <sup>a***</sup>	.667 (.272) <sup>b*</sup>	.381 (.334) <sup>b*</sup>
LFSW	-.058 (.321)	-.371 (.504)	.443 (.377)	.477 (.341)

\* $p < 0.05$ ; \*\*\* $p < 0.001$

#### 8.4.6 Relationship between energy intake measured in the laboratory and self-reported free-living energy intake

Table 8.9 shows that overall energy intake from the TM-EI and the DR-EI were positively correlated. Additionally, snack intake from the DR-EI was positively related to snack intake in the laboratory and to overall energy intake from both the TM-EI and the DR-EI.

Table 8.9 Pearson's correlations between energy intake (kcal) measured in the laboratory and self-reported free-living energy intake.

		Dietary recall day				
		Overall	Breakfast	Lunch	Dinner	Snacks
Laboratory energy intake	Overall	.585***	.117	.103	.302	.419**
	Breakfast	.331*	.048	.112	.247	.148
	Lunch	-.034	-.167	-.079	.351*	-.233
	Dinner	.678***	.265	.260	.186	.490**
	Snacks	.391**	.067	-.034	.101	.434**

\* $p < 0.05$ ; \*\* $p < 0.01$ ; \*\*\* $p < 0.001$

#### **8.4.7 The relationship between food hedonics and free-living energy intake and macronutrient choice**

##### **8.4.7.1 Energy intake**

There were no relationships between explicit liking or explicit wanting and self-reported overall energy intake in the fasted or fed state. When fasted, implicit wanting for LFSA was negatively associated with overall self-reported energy intake [ $r(34) = -.461, p < 0.01$ ] and self-reported snack food intake [ $r(34) = -.386, p < 0.02$ ]. Further to this, implicit wanting for high fat sweet foods assessed in a fasted state was associated with number of sweet snack foods consumed [ $r(34) = .378, p < 0.03$ ]. There were no relationships between measures of implicit wanting in the fed state and self-reported energy intake.

##### **8.4.7.2 Macronutrient choice**

Implicit wanting for LFSA was negatively related to total fat intake [ $r(34) = -.588, p < 0.001$ ] and total carbohydrate [ $r(34) = -.389, p < 0.02$ ]. Additionally there was a negative relationship between liking for LFSA and fat intake [ $r(34) = -.342, p < 0.05$ ].

### **8.5 Discussion I**

The current study examined food intake in the laboratory and under free living conditions over two 24-hour periods in order to determine whether the previous laboratory-based findings in the current thesis which suggest that greater binge eating severity is related to increased consumption of, and an enhanced preference for high-fat sweet foods extend to free-living eating behaviour. Additionally, the current study explored whether measures of liking and wanting, assessed by the LFPQ, were related to free-living, self-reported eating behaviour.

There was no effect of trait binge eating on ratings of appetite sensations or on the satiating efficiency of food. During the test meal methodology energy intake day (TM-EI), O-B consumed more energy from the ad libitum dinner, and more energy from the snack box compared to O-NB. These findings are consistent with previous

research that has shown individuals with the propensity to binge eat tend to consume a greater number of calories in the second half of the day (Allison & Timmerman, 2007; Raymond et al., 2003). It may be that in the current study the introduction of the snack box following lunch challenged O-B's earlier attempts to restrict or 'be good' (consistent with the literature examining the relationship between dietary restriction and binge eating e.g. (Abbott et al., 1998; Goldschmidt, Wall, Loth, Le Grange, & Neumark-Sztainer, 2012) and once they had given in such attempts to restrict or control their intake were no longer made. This suggestion is consistent with the trend that O-B reported consuming more energy during the evening of the TM-EI compared to O-NB and with the finding that O-B reported experiencing greater cravings for palatable foods. Conversely, it may be that during the second half of TM-EI period there were more opportunities for food intake to vary due to the greater amount of food provided.

In accordance with the findings from Chapter 7, O-B exhibited a strong preference for, and increased consumption of sweet foods, especially high fat sweet foods, in the snack box, and reported experiencing more intense food cravings compared to O-NB. One of the aims of the current study was to assess whether O-B reduce their subsequent intake to account for the extra calories consumed from snack foods. More specifically, it aimed to assess whether O-B over-consumed over the course of the day. To define and analyse overconsumption, an estimate of participants' individual energy requirements were calculated and compared to their actual daily energy intake. Compared to O-NB, O-B consumed more energy during the first 24-hour period than their estimated daily energy requirements indicating that they had over consumed. Interestingly, however, there was evidence for overconsumption in all groups during TM-EI, with overconsumption being the lowest in O-NB (L-B and L-NB over consumed by 41% and 25%, respectively, while O-B and O-NB over consumed by 34% and 6%, respectively). The overconsumption observed in L-B appears to have been driven by a large number of calories consumed at the test meal

lunch and from snack food intake. It does seem that they may have attempted to reduce their intake later in the day as they consumed less from the test meal dinner than L-NB (albeit not significantly less). Similarly, L-NB consumed an almost equal amount of calories from lunch and dinner suggesting that the test meal lunch may have facilitated consumption, perhaps due to the increased degree of variety that was offered at this meal compared to breakfast and dinner.

A similar pattern of overconsumption was not observed from the DR-EI, during which self-reported energy intakes were more in line with estimated daily energy requirements, especially for both lean groups. These disparities between intakes may have arisen for several reasons. Firstly, the current study provided all test meals in ad libitum quantities and within each test meal there were a few items that participants could choose from. Therefore, these findings may suggest that this type of laboratory study is perhaps conducive to increasing consumption beyond what an individual would habitually consume. For example, Hetherington et al. (2006) found that providing participants with a variety of food stimulates intake by delaying the development of satiation. Furthermore, participants in the current study were instructed to eat until they felt comfortably full which previous research suggests may also increase energy intake beyond what would have been consumed if participants were asked to eat until the pleasantness of the food declined (Poothullil, 2002). Secondly, it may be that the increased intake observed in the TM-EI period had an impact on food intake the following day, reducing it below what it normally would be. In order to address this, future research should ideally conduct two or three dietary recall procedures following the initial 48-hour test period in order to obtain an average of free-living intake that is more certainly unaffected by any laboratory procedures. Finally, dietary recall procedures are vulnerable to high rates of underreporting partly due to difficulties in remembering dietary details and partly due to embarrassment regarding how much was eaten. The likelihood of underreporting increases with BMI and may account for the low self-reported intake

of O-NB (Moshfegh et al., 2008). However, while not significantly different, O-B did report consuming a large proportion of calories from snack foods compared to O-NB. When the types of snack foods were examined the increased preference for sweet foods observed in the laboratory was evident in their self-reported free living eating behaviour supporting the notion that trait binge eating is associated with an enhanced preference for sweet foods. Further to this, the enhanced preference for sweet foods was evident not only in snack food intake but also within meals in the dietary recalls of O-B (see Appendix 7 for an example of three O-B dietary recalls).

When the associations between laboratory eating behaviour and free living eating behaviour were explored there was a good association between overall energy intake assessed under laboratory and free-living conditions. Promisingly, there was a strong relationship between free-living snack intake and laboratory snack intake, which supports the validity of the ad libitum eating task used in the experimental chapters of the current thesis.

Interesting group differences emerged with regards to measures of body adiposity as O-B had a greater amount of fat mass and a trend towards a larger waist circumference than O-NB. The increase in waist circumference was also found in Chapter 7 and may indicate an elevated risk of obesity-related health problems (Zhu et al., 2002). One early investigation into binge eating in the obese found that the likelihood of binge eating increased with adiposity, which was assessed using the BMI (Telch, Agras, & Rossiter, 1988).

In addition to examining food intake and food choice the current study also investigated whether there were any self-reported differences in mood throughout the TM-EI and for the preceding seven days using the Mood subscale from the Control of Eating Questionnaire (COEQ; Hill, Weaver & Blundell, 1991). Over the course of the TM-EI there was a trend for levels of contentedness to be lower in O-B compared to O-NB. This finding resonated with the outcome from the Positive Mood subscale of the COEQ for which O-B scored lower than O-NB suggesting that they



experience lower levels of positive mood outside of the laboratory as well as within. This finding is in line with the outcome of Chapter 7, and previous research that has shown individuals with subclinical levels of binge eating tend to report experiencing low mood (Goldschmidt, Tanofsky-Kraff, & Wilfley, 2011; Goldschmidt et al.; Greeno et al., 2000; Wegner et al., 2002). Greeno et al. (2000) reported that females without BED but who engaged in binge eating experienced lower mood than those without BED who did not engage in binge eating. However, Wegner et al. (2002) found no evidence that binge eating was a response to a low mood state or that binge eating provided relief from negative mood which suggests that low mood may be a characteristic of individuals with the tendency to binge eat rather than solely being related to binge eating behaviour itself.

Consistent with the findings from Chapter 7, the current chapter demonstrated that higher trait binge eating scores were associated with enhanced liking for foods, in particular high fat foods and enhanced implicit wanting for high-fat sweet foods that was independent of motivational state. O-NB responded faster for savoury foods in the fasted state and for sweet foods in the fed state. These findings are in contrast to O-NB examined in Chapter 7. However, it is important to note in the present study participants were recruited on the basis of their binge eating score. To this end, the findings with regards to those scoring high on the BES are more likely to be consistent whereas the findings in those scoring low on the BES would perhaps be less expected to be consistent as they may be less likely to share common characteristics, other than having a low binge eating score which would not help to explain the aetiology of obesity in these individuals. Therefore, there is likely to be a higher degree of heterogeneity amongst O-NB, perhaps without one common risk factor to account for why they are overweight or obese. This may in part account for the differences observed for O-NB outcomes in this thesis, compared to the relative consistency observed in O-B individuals.

Finally, the current study examined whether the measures of liking and wanting were related to free-living eating behaviour. It was demonstrated that implicit wanting for low-fat savoury foods was negatively associated to overall self-reported energy intake and self-reported snack food intake. Further to this, implicit wanting for high-fat sweet foods was positively associated with the number of sweet snack foods consumed. Enhanced implicit wanting for low-fat savoury foods may be protective against overconsumption and this may be reflected through healthier diet choices. This suggestion is supported by the positive relationship between implicit wanting for low-fat savoury and liking for low-fat savoury and their association with lower fat intake.

## 8.6 Results II

### 8.6.1 Food addiction symptoms

Table 8.10 shows that O-B had a greater number of food addiction symptoms on the YFAS compared to O-NB [ $t(22) = 3.69, p < 0.001$ ]. There were no differences between the lean groups. The frequencies for each symptom endorsed by each group indicated that all participants felt that they had problems cutting down on palatable foods. Additionally, a greater number of O-B seemed to report experiencing more addictive-like behaviours towards palatable foods.

Table 8.10 Mean (standard deviation) YFAS symptom count for O-B, O-NB, L-B and L-NB and the number of participants in each group endorsing each symptom

	O-B (n=12)	O-NB (n=12)	L-B (n=5)	L-NB (n=5)
Overall symptoms	3.75 (2.3)	1.25 (.452)	2.00 (.707)	2.60 (2.61)
Withdrawal	5	1	1	1
Tolerance	2	0	2	1
Sustained use	8	1	0	1
Activities	6	0	2	1
Time	6	0	0	2
Loss of control	5	1	1	2
Problems cutting down	12	12	5	5

### 8.6.2 Food addiction diagnosis

Using the YFAS diagnostic scoring procedure, four out of the twelve individuals in the O-B group met the criteria to be classified as a ‘food addict’ according to Gearhardt et al. (2009). None of the individuals in O-NB, L-B or L-NB met the criteria for food addiction therefore the subsequent analyses will focus on comparisons between individuals within O-B classified as either ‘food addicts’ (n = 4) or ‘non-food addicts’ (n = 8).

### 8.6.3 Characteristics of ‘food addicts’ compared to ‘non-food addicts’

Table 8.11 shows that O-B ‘food addicts’ had a larger waist circumference and greater percentage body fat compared to O-B ‘non-food addicts’. Additionally, ‘food addicts’ scored higher on TFEQ disinhibition and trait binge eating compared to ‘non-food’ addict while the difference in TFEQ restraint approached significance [ $t(10) = 2.09$ ,  $p=0.06$ ].

Table 8.11 Mean (standard deviation) age, anthropometric, body composition and psychometric trait characteristics of O-B classified as ‘food addicts’ and ‘non-food addicts’

	Food addict (n=4)	Non-food addict (n=8)
Age	26.75 (9.91)	25.13 (6.33)
BMI (kg/m <sup>2</sup> )	34.16 (5.36)	30.14 (3.93)
Fat mass (kg)	46.43 (12.15)*	31.22 (11.07)*
Body fat (%)	45.90 (5.88)*	35.96 (8.04)*
Fat free mass (kg)	53.68 (5.93)	53.98 (4.18)
Waist (cm)	109.06 (10.52)*	92.57 (11.75)*
Restraint	14.25 (2.87)	9.38 (4.14)
Disinhibition	14.75 (1.25)*	10.75 (3.24)*
Hunger	11.25 (1.50)	8.25 (3.92)
Binge eating score	23.50 (2.38)*	19.88 (2.53)*

\* $p<0.05$

### 8.6.4 Experience of food cravings

‘Food addicts’ scored significantly higher on the Craving Intensity [ $t(11) = 2.56$ ,  $p<0.05$ ] and significantly lower on the Mood [ $t(11) = 3.28$ ,  $p<0.01$ ] subscales of the

COEQ (see Table 8.12). The difference in Craving for Sweet Foods approached significance [ $t(11) = 2.12, p=0.06$ ].

Table 8.12 Mean (standard deviation) Craving Intensity, Mood, Craving for Savoury, Craving for Sweet and Feelings of Fullness subscales of COEQ for O-B classified as ‘food addicts’ and ‘non-food addicts’

	Food addict (n=4)	Non-food addict (n=8)
Craving Intensity	77.05 (15.34)*	54.77 (13.12)*
Mood	33.75 (9.54)**	57.14 (12.18)**
Craving for Savoury	60.88 (20.91)	50.57 (20.96)
Craving for Sweet	80.15 (10.11) <sup>1</sup>	56.66 (20.48) <sup>1</sup>
Feelings of Fullness	51.75 (24.31)	61.00 (24.41)

\* $p<0.05$ ; \*\* $p<0.01$ ; <sup>1</sup> $p=0.06$

### 8.6.5 Test meal methodology energy intake

Under laboratory conditions, and when accounting for differences in energy requirements, ‘food addicts’ consumed more overall energy [ $F(1, 9) = 5.13, p<0.05$ ] and more energy from snack foods [ $F(1, 9) = 9.59, p<0.01$ ] compared to ‘non-food addicts’ (Table 8.13). When the types of snack food consumed were explored (Figure 8.9) an interaction between fat content and food addict group revealed that ‘food addicts’ consumed more high fat snack foods compared with ‘non-food addicts’ [ $F(1, 10) = 8.46, p<0.02$ ].

Table 8.13 Mean (standard deviation) energy intake (kcal) during TM-EI for O-B classified as ‘food addicts’ and ‘non-food addicts’

	Food addict (n=4)	Non-food addict (n=8)
Overall energy intake	3485.66 (438.31)*	2787.87 (514.17)*
Breakfast	542.29 (170.76)	520.68 (179.07)
Lunch	767.33 (132.52)	828.12 (185.93)
Dinner	1101.98 (212.79)	883.63 (261.22)
Overall snack intake	1075.07 (388.99)**	555.45 (170.32)**

\* $p<0.05$ ; \*\* $p<0.01$

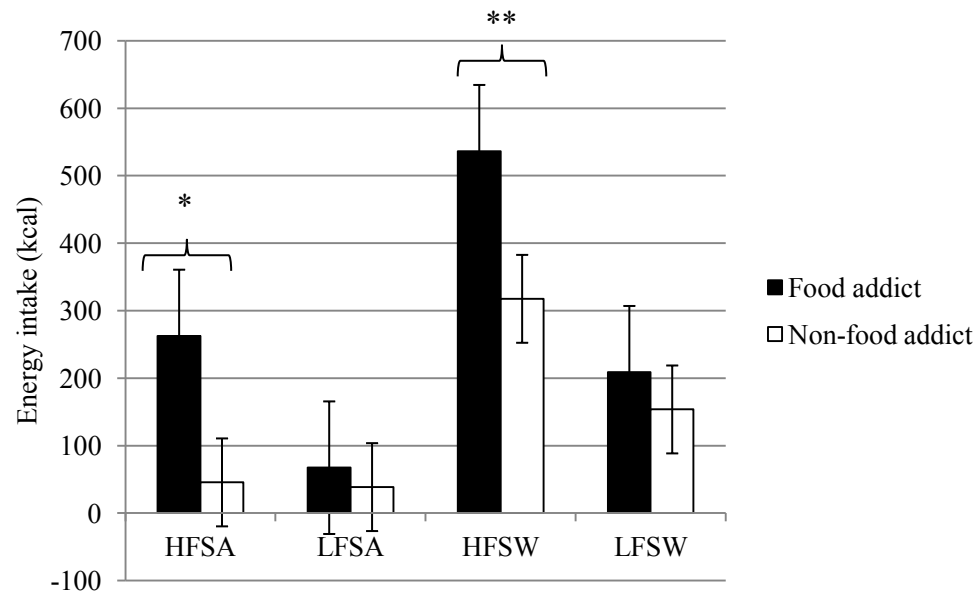


Figure 8.9 Energy intake (kcal) according to fat content and taste of the snack foods for O-B classified as 'food addicts' and 'non-food addicts'

\* $p < 0.05$ ; \*\* $p < 0.01$

### 8.6.6 Dietary recall self-reported energy intake

Under free-living conditions, 'food addicts' reported consuming more overall energy compared to 'non-food addicts' (see Table 8.14). In addition, 'food addicts' consumed more calories from dinner and snack foods compared to 'non-food addicts', however only the latter finding was significantly different [ $F(1, 9) = 8.37$ ,  $p < 0.02$ ].

Table 8.14 Mean (standard deviation) self-reported energy intake during the dietary recall day for 'food addicts' and 'non-food addicts'

	Food addict (n=4)	Non-food addict (n=8)
Overall energy intake	3128.74 (517.07)	2481.94 (614.84)
Breakfast	470.30 (304.89)	555.14 (160.45)
Lunch	604.33 (113.50)	699.10 (210.04)
Dinner	1047.86 (417.30)	787.45 (339.88)
Overall snack intake	1006.25 (408.88)*	440.26 (223.21)*

\* $p < 0.05$

## 8.7 Discussion II

The second aim of the current study was to examine whether the construct of 'food addiction' as defined by the Yale Food Addiction Scale (Gearhardt et al., 2011)

formed part of a distinct subtype in overweight or obese individuals with moderate levels of binge eating.

O-B endorsed more food addiction symptoms compared to O-NB suggesting that O-B may experience more addiction like tendencies towards food. To explore this further, O-B were divided into those who met, and those who did not meet the diagnostic threshold for food addiction according to Gearhardt et al., (2011). To meet the diagnostic threshold three or more symptoms must be endorsed in addition to the experience of significant impairment or distress from eating behaviour. Four individuals in O-B met this threshold and these were used to create the 'food addict' group while the remaining eight formed the 'non-food addict' group.

Using the newly defined groups, it was shown that 'food addicts' had a larger waist circumference, and greater levels of fat mass and percentage body fat compared to 'non-food addicts' with no corresponding difference in BMI. To date there does not appear to be any corroborating evidence for this finding and therefore further research is needed for confirmation. In addition, 'food addicts' scored higher on trait disinhibition and trait binge eating suggesting that 'food addicts' may experience a greater level of eating pathology compared to 'non-food addicts'. This finding is in line with greater eating pathology reported in previous research examining obese individuals with BED who also met the criteria for food addiction (Davis, Curtis, et al., 2011; Gearhardt et al., 2013; Gearhardt et al., 2011).

Furthermore, the current study demonstrated that 'food addicts' consumed more energy overall, and more energy from snack foods during TM-EI compared to 'non-food addicts'. This finding was replicated in the self-reported energy intake from DR-EI as 'food addicts' reported consuming significantly more energy from snack foods compared to 'non-food addicts' and there was a trend for the 'food addicts' to report consuming more energy overall throughout the course of the day. Interestingly, the 'food addicts' were, at least in part, distinguishable from the overall O-B in their choice of snack foods from the snack box provided during TM-

EI, as they consumed more energy from high-fat snack foods rather than showing a strong preference for high-fat sweet foods specifically. Taken together, these findings suggest that 'food addiction' as defined by the YFAS, appears to fit along the continuum of the BES and may correspond to a more severe expression of binge eating tendency that is characterised by greater levels of eating pathology, greater adiposity, enhanced cravings for food, and greater snack food consumption. Nevertheless, there are strong social and political implications in adopting the term 'food addict' in scientific research. These preliminary results suggest the psychometric construct of 'food addiction' may not be behaviourally distinct from binge eating tendency. Therefore, the classification of certain individuals as 'food addicts' may not be appropriate.

## **8.8 General Discussion**

The first aim of the current study was to determine whether our previous findings in overweight or obese individuals with high or low levels of trait binge eating extended beyond the laboratory situation and related to eating behaviour in the natural setting. Consistent with the previous chapters, it was demonstrated that higher trait binge eating scores were associated with enhanced craving intensity and cravings for sweet foods, increased intake of high-fat sweet foods, increased liking for food overall and enhanced implicit wanting for high-fat sweet foods that was independent of motivational state.

Results from DR-EI complemented the laboratory-based observations as O-B reported consuming a greater number of sweet snack foods compared to O-NB. In addition, there was a trend for sweet snack preference in the free-living eating behaviour of L-B. The association between laboratory-based and free-living based measures of eating behaviour were good, especially for snack food intake. These findings help to validate the ad libitum eating task used in the current thesis as a sensitive measure of energy intake and food preferences. In addition, our previous findings were extended as the current study demonstrated that O-B consumed a

greater amount of energy from fat and saturated fat compared to O-NB during DR-EI indicating that dietary recall procedures are able to supplement laboratory findings, where variation in macronutrient content is less able to vary.

The second aim of the current study was to explore the recent idea that ‘food addiction’ may form a subtype of disordered eating distinct from binge eating tendency. In line with previous research in BED (Davis, Curtis, et al., 2011; Gearhardt et al., 2013; Gearhardt et al., 2011), the current study found that the YFAS criteria for food addiction identified the higher scores within the obese binge eating subtype and was associated with increased levels of eating pathology, body adiposity, craving for food and energy intake. However, it is important to note that these findings should be considered with caution due the small sample examined in the current study and the very low number of individuals meeting the YFAS criteria for food addiction.

The present study had some limitations that should be considered. Firstly, the 24-hour dietary recall procedure would have been more reliable if conducted on at least three different occasions in line with previous research (Epstein et al., 2011; Moshfegh et al., 2008; Stote, Radecki, Moshfegh, Ingwersen, & Baer, 2011) in order to gather enough information to create a more valid estimate of free-living eating behaviour. However, a strength of the current study was that we controlled for weekend fluctuations in energy intake by only holding the dietary recall days on Wednesdays and Thursdays. Secondly, as participants were not told that binge eating behaviour was being assessed it was not possible to determine, in the free-living energy intake data, whether periods in which a large amount of food was consumed could be classified as a binge. Previous research has used participants’ self-reports of when a binge occurred (by asking whether there was a loss of control) alongside more objective energy intake cut off points (e.g. situations where greater than a 1000 calories are consumed) to determine what foods are binged on and what factors precede bingeing behaviour (Allison & Timmerman, 2007; Raymond et al., 2003).



Thirdly, future research should perhaps conduct the dietary-recall day one week after the laboratory-based measures of energy intake to avoid any overconsumption that occurs in the laboratory from influencing what is consumed the next day. Finally, it is likely that the null outcomes with regards to differences between L-B and L-NB are the result of the low number of participants in these groups. It would be worthwhile to further examine free-living eating behaviour in a larger number of L-B and L-NB. It should be noted that the inclusion of four participants who had also participated in the study presented in Chapter 7 might have resulted in an increased likelihood of demand characteristics in these individuals. However, the debriefing procedure for the study presented in Chapter 7 did not allude to the specific aims of the thesis but rather presented the study as an investigation into individual differences in the responsiveness to food cues (see Appendix 8 for the debriefing statement). Therefore, these participants were unaware that the aim of the study was to examine individual differences in trait binge eating or that we had previously found that trait binge eating was associated with increased preference for high-fat sweet foods.

In summary, the current study extended the previous findings in this thesis by showing that the binge eating subtype has relevance for free-living eating behaviour assessed by 24-hour dietary recall. In addition, individuals meeting the criteria for food addiction as defined by the YFAS, appear to correspond to the highest BES scores within the O-B group

## **8.9 Summary**

- Higher levels of binge eating severity were associated with increased preference for sweet snack foods in both laboratory-based and free-living based measures of eating behaviour.
- Consistent with previous chapters, the current chapter demonstrated that greater levels of binge eating severity was associated with increased levels of adiposity, enhanced craving intensity and greater cravings for sweet foods, increased

intake of high-fat sweet foods, increased liking for food overall and enhanced implicit wanting for high-fat sweet foods independent of motivational state.

- Four of the O-B met the criteria for 'food addiction'. These individuals exhibited particularly high binge eating scores, greater levels of eating trait pathology, increased craving for food, greater energy intake and different food preferences compared to those in the O-B group who did not meet the criteria for 'food addiction'.

## Chapter 9

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### **Examination of potential genetic markers for the trait binge eating phenotype and its intermediary characteristics**

#### **9.1 Abstract**

The current study had two aims: 1) to determine whether a common underlying genotype could be identified for the trait binge eating phenotype that has been characterised in the previous chapters of this thesis; and 2) to examine the associations between relevant common gene variants and the characteristics that have been identified to be associated with trait binge eating. One hundred and eighty participants (BMI:  $23.5 \pm 3.8 \text{ kg/m}^2$ ; Age:  $26.1 \pm 9 \text{ yrs}$ ) were recruited from two areas of the UK following identical protocols and procedures. Participants attended their respective research units on two occasions. On the first visit, participants arrived in the morning following an overnight fast so that anthropometric measures, including body composition could be taken. On the second visit, participants arrived at lunchtime following a 3.5-hour fast and completed the LFPQ and the ad libitum eating task in a fasted state. DNA was isolated from saliva and genotyped for candidate genes that were identified on the basis of a literature search to find relevant single nucleotide polymorphisms (SNPs) that have previously been or were hypothesised to be associated with binge eating, preferences for foods high in sugar or fat, and those related to obesity. The genotype frequency of the examined SNPs did not differ between high and low scorers on the Binge Eating Scale. This suggests that there was no common genotype underlying the trait binge eating phenotype. The examination of the intermediary phenotypes revealed that the rs6277 polymorphism in the dopamine D2 receptor gene (DRD2), the rs5400 polymorphism of the SLC2A2 gene and the rs9939609 and rs1121980 polymorphisms in the FTO gene were associated with energy intake and food choice. The rs1800497 polymorphism of the DRD2 associated ANKK1 gene, the DRD2 rs6277 polymorphism, the rs35874116 polymorphism of the sweet taste receptor TAS1R2 gene and the rs2151916 and

rs1761667 polymorphisms in the CD36 gene were associated with anthropometrics and body composition. In addition, ANKK1 rs1800497 was associated with reward responsiveness. Furthermore, the SLC2A2 rs5400 polymorphism was associated with food liking. These findings are discussed with reference to relevant theories and in light of the study's limitations.

## **9.2 Introduction**

The previous chapters in this thesis have provided evidence to support that trait binge eating forms part of a hedonic phenotype of obesity that is characterised by differences in energy intake, food choice, macronutrient preferences, liking and wanting, body composition and experiences of food craving. The first aim of the current chapter was to examine whether an underlying common genotype could be identified for this behavioural phenotype using a candidate gene approach. The second aim was to examine associations between common gene variants and the characteristics that have been previously identified to be associated with trait binge eating (so-called 'intermediary phenotypes'). Candidate genes were identified on the basis of a literature search to find relevant SNPs that have previously been or were hypothesised to be associated with binge eating; preferences for foods high in sugar or fat; and those related to obesity.

### **9.2.1 Reward related genes**

Previous research examining genetic markers of Binge Eating Disorder (BED) have primarily focused on SNPs within genes that encode for reward related neuropeptides. Most commonly studied are the mu opioid receptor gene (OPRM1) and genes that are related to, or encode for the dopamine D2 receptor (DRD2). Through the examination of several common variants in these genes, it has been proposed that BED is a distinct, biologically based, subtype of obesity that is characterised by enhanced dopamine and opioid functionality and hyper-responsiveness to palatable foods (Davis et al., 2009; Davis et al., 2012).

### **9.2.1.1 Opioid related genes**

Since their discovery in the 1970s, research has frequently shown that opioid peptides are involved in ingestive behaviour with a specific role in mediating the orosensory reward properties of food. In humans, opioid antagonists, such as naloxone and naltrexone, have reliably been shown to reduce the hedonic response to, and intake of, palatable foods (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1995; Yeomans & Gray, 2002; Ziauddeen et al., 2012) and have been shown to reduce binge-eating behaviour in rats (Boggiano et al., 2005). Drewnowski et al. (1995) found that naloxone reduced the hedonic ratings of varying sugar and fat dairy mixtures but did not alter the judgement of fat content or the perception of sweetness in a sample of females with and without binge eating tendencies. In addition, they found that naloxone significantly reduced energy intake in all participants. Interestingly, however, in those with binge eating tendencies, the effect of naloxone was specific for high-fat sweet foods. More recently, Ziauddeen et al. (2012) evaluated the effect of a novel opioid antagonist on eating behaviour and hedonic responses to sweetened dairy products in a sample of obese individuals who experienced problems with binge eating. In line with Drewnowski et al. (1995), they found that the antagonist reduced hedonic ratings for the dairy products high in sugar and fat. Furthermore, the antagonist specifically reduced the intake of dessert foods in an ad libitum buffet test meal.

The OPRM1 gene has been extensively studied for its role in substance abuse, including alcohol and heroin (Bart et al., 2004; Drakenberg et al., 2006; Szeto, Tang, Lee, & Stadlin, 2001). One commonly investigated SNP in the OPRM1 gene is rs1799971 (commonly referred to as A118G). The functional relevance of this SNP is not agreed upon with conflicting evidence in the literature that supports both a loss-of-function (Kroslak et al., 2007; Zhang, Wang, Johnson, Papp, & Sadée, 2005) and a gain-of-function (Bond et al., 1998) for the minor G allele. Another, less commonly investigated SNP in the OPRM1 gene is rs495491 of which the functional

significance is currently unknown. However, it has been suggested that the C-allele may be associated with reduced opioid receptor activity (Shabalina et al., 2009). Davis, Zai, et al. (2011) examined whether variance in rs1799971 and rs495491 was associated with self-reported food preferences (assessed using Food Preference Questionnaires) in a non-clinical population. They found that individuals homozygous for the G-allele of rs1799971 had a stronger preference for high fat and high sugar foods compared to the other genotypes, whereas individuals homozygous for the C-allele of rs495491 reported a lower preference for sugar and fat (Davis, Zai, et al., 2011). Based on the previous research outlined, it was hypothesised that variation at rs1799971 and rs495491 would be associated with an increased preference for high-fat sweet foods and an increased liking for high-fat foods.

#### **9.2.1.2 Dopamine related genes**

The degree of pleasure from eating has been shown to correlate with the quantity of dopamine released within the brain in both humans (Small, Jones-Gotman, & Dagher, 2003) and non-human animals (Hernandez & Hoebel, 1988). Individual variability in the level of dopamine available has been researched extensively in the addiction literature. Two theories have emerged with regards to the role dopamine may have in the development of addiction disorders and obesity. The first favours the view that a hypo-functioning reward system is a key contributor, in which individuals are posited to have an innate low reward response and therefore increase their use of rewarding substances in order to obtain an 'optimal' level of reward (Comings & Blum, 2000; Geiger et al., 2009). The second favours the view that a hyper-responsive reward system results in an enhanced motivation to seek out rewarding substances (Berridge et al., 2010; Davis et al., 2007). In an attempt to resolve the opposing views some authors have suggested that a hypo-functioning reward system may be a consequence and not a cause of obesity in which the dopamine receptors have been down regulated in response to excessive activation (Berridge et al., 2010; Davis et al., 2004; Steele et al., 2010). Partial support for this

suggestion was demonstrated by Steele et al. (2010) who compared dopamine D2 receptor binding in obese individuals before and after gastric bypass surgery. They found that six weeks post-surgery there was an increase in dopamine D2 receptor availability that was proportional to the amount of weight lost. Furthermore, post-surgery there were no differences in dopamine D2 receptor binding between the gastric bypass patients and non-obese controls suggesting that the lower level of D2 receptor availability pre-surgery had been reversed. In a prospective fMRI study, Stice, Yokum, Blum, et al. (2010) examined whether weight gain was associated with reduced striatal activation in response to palatable food intake in a sample of obese women. They found that compared to weight stable women, those who gained weight over a six-month period had lower striatal activation in response to palatable food intake relative to baseline response.

The DRD2 gene encodes the dopamine D2 receptor. Two commonly investigated SNPs that are within or associated with the DRD2 gene are rs6277 (commonly referred to as C957T) and rs1800497 (commonly referred to as Taq1A). rs1800497 was previously believed to be located in the 3'-untranslated region of DRD2 (Noble, 2000), however Neville, Johnstone, and Walton (2004) demonstrated that rs1800497 actually resides in a nearby gene, Ankyrin Containing Kinase 1 (ANKK1). The presence of the A1 allele of rs1800497 has been associated with a 30-40% reduction in the number of dopamine D2 receptors and weaker dopamine signalling (Noble et al., 1991; Ritchie & Noble, 2003; Thompson et al., 1997). Variation at rs6277, which is in linkage disequilibrium with rs1800497, has been associated with a dose dependent effect on dopamine binding potential with C-allele homozygotes having the lowest binding, heterozygotes having intermediate binding and T-allele homozygotes having the highest binding (Hirvonen et al., 2004; Hirvonen et al., 2005).

Some reports have suggested that the presence of the A1 allele is associated with an increase in BMI (Blum et al., 1996; Chen et al., 2012), however evidence appears to

be mixed (Jenkinson et al., 2000). The A2/A2 genotype of rs1800497 has been implicated in BED. Evidence suggests that BED is associated with a greater density of D2 receptors and higher D2 binding potential, which is consistent with the notion that BED is characterised by hyper-responsiveness to reward (Davis et al., 2009; Davis et al., 2012). Davis et al (2012) examined five functional markers of the DRD2 gene, including rs1800497 and rs6277, in a sample of overweight or obese males and females with and without BED. They found that a greater number of individuals with BED were homozygous for the A2 allele of rs1800497 and homozygous for the T allele of rs6277 compared to obese controls. Additionally, individuals with the A2/A2 genotype had significantly higher binge eating scores, and reported experiencing greater food cravings compared to those with at least one copy of the A1 allele (Davis et al., 2012). Therefore, these findings suggest that binge eating may be associated with hypersensitivity rather than hyposensitivity to reward.

#### **9.2.1.2.1 Binge eating disorder: Joint action of opioid and dopamine related genes**

The notion that BED is characterised by a hyper-functioning reward system is further supported by studies that examine the combined influence of genetic variation in the OPRM1 gene and the DRD2 associated ANKK1 gene. Davis et al. (2009) examined psychological and genetic markers of hedonic eating in individuals with and without BED. Participants were genotyped for the OPRM1 rs1799971 and the ANKK1 rs1800497 polymorphisms. It was demonstrated that a greater proportion of individuals with BED had the A2/A2 genotype of rs1800497 and at least one copy of the rs1799971 G allele – both variants are associated with greater functionality. Taken together, these findings suggest that the tendency to binge eat may be underpinned by genetically based hyper-responsiveness to the hedonic properties of food.



### **9.2.2 Taste related genes**

The sensory aspects of food are a powerful contributor to dietary preferences, and taste is an important determinant of food selection (Drewnowski, Henderson, & Barratt-Fornell, 2001; Glanz, Basil, Maibach, Goldberg, & Snyder, 1998). The genes that encode four of the five basic taste modalities have been identified and include the TAS1R gene family for sweet and umami taste (Bachmanov et al., 2011; Zhao et al., 2003), the TAS2R gene family for bitter taste (Adler et al., 2000; Chandrashekar et al., 2000) and the PKD2L1 and PKD1L3 genes for sour taste (Huang et al., 2006; Ishimaru et al., 2006; Kataoka et al., 2008). To date, the genes underlying salt taste are not well understood (Kim, Breslin, Reed, & Drayna, 2004). More recently, the CD36 gene has been proposed as a potential fat taste receptor (Laugerette et al., 2005; Martin et al., 2011; Mattes, 2009). Variation in the genes that encode for taste receptors may contribute for differences in food preferences and dietary choices, and may be associated with the enhanced preference for high-fat sweet foods observed in individuals with the propensity to binge eat.

#### **9.2.2.1 Genes for sweet taste**

The TAS1R gene family is comprised of three members; TAS1R1, TAS1R2 and TAS1R3. TAS1R3 is often co-expressed in the taste receptor cells with TAS1R1 and TAS1R2, to form the taste receptors for umami taste (TAS1R1+3) and sweet taste (TAS1R2+3) (Li, 2009; Nelson et al., 2001). Of particular interest to the current study is the sweet taste receptor TAS1R2+3. Previous research has shown that the rs35874116 variant (commonly referred to as Ile191Val) in the TAS1R2 gene is associated with the habitual intake of sugar in overweight and obese young adults, with individuals homozygous for the T allele consuming more sugar in their self-reported daily diet compared to heterozygotes or those homozygous for the C allele (Eny, Wolever, Corey, & El-Sohemy, 2010). Furthermore, Fushan, Simons, Slack, Manichaikul, and Drayna (2009) examined two variants in TAS1R3 gene (rs307355 and rs35744813) and demonstrated that for both SNPs, individuals who carried the T

allele had reduced sensitivity to sucrose solutions in a dose dependent manner compared to individuals who were homozygous for the C allele. Consistent with this phenotype, the authors showed that the T allele of each SNP resulted in reduced promoter activity compared to the C allele suggesting a functional mechanism for this effect.

The SLC2A2 gene encodes for the GLUT2 member of the facilitative glucose transport family and is expressed in the intestine, pancreas, kidney, liver and brain (Bell et al., 1990; Fukumoto et al., 1989; Thorens, Cheng, Brown, & Lodish, 1990). Studies in non-human animals have shown that compared to wild-type mice, GLUT2 knockout mice consume 27% more energy (Bady et al., 2006). To examine whether this increase in intake was due to impaired glucose sensing, Bady et al. (2006) administered injections of glucose. They found that in the GLUT2 knock-out mice, feeding was not inhibited by glucose, and unlike the wild-type mice, there was no decrease in NPY or increase in POMC expression observed, suggesting that GLUT2 acts as a glucose sensor that regulates the expression of neuro-peptides (Bady et al., 2006). In humans, genetic variation in SLC2A2 has been linked to increased habitual sugar intake (Eny, Wolever, Fontaine-Bisson, & El-Sohemy, 2008). Eny et al. (2008) examined the rs5400 SNP (commonly referred to as Thr110Ile) in the SLC2A2 gene in a healthy young adult sample, and in a sample of individuals with Type 2 diabetes. In both samples, the presence of the T allele was associated with increased habitual intake of sugar, specifically the increased intake of sucrose, fructose and glucose. These findings suggest that genetic variation in TAS1R2, TAS1R3 and SLC2A2 may be associated with individual differences in the preference for sugar-containing foods and beverages and may therefore be connected with the increased preference for sweet foods observed in trait binge eating.

#### **9.2.2.2 Genes for bitter taste**

The TAS2R gene family is much larger than the TAS1R family, comprising of twenty-five bitter taste receptor genes (Tepper, 2008). The TAS2R38 gene controls

sensitivity to the bitter taste of 6-*n*-propylthiouracil (PROP) and phenylthiocarbamide (PTC) (Kim et al., 2003). Three SNPs (rs713598, rs1726866 and rs10246939) in the TAS2R38 gene contribute to the likelihood that an individual will be either a taster or a non-taster of PROP (Tepper, 2008). Research has found that an inverse relationship between the preference for high fat foods and for sweet foods and the perception of bitter taste (Duffy & Bartoshuk, 2000; Tepper & Nurse, 2006). Furthermore, studies have shown that PROP supertasters were more likely to be 'sweet dis-likers' compared to non-tasters (Looy & Weingarten, 1992; Yeomans, Tepper, Rietzschel, & Prescott, 2007) suggesting that PROP taster status is linked to a preference for sweet foods.

### **9.2.2.3 Genes for fat taste**

Fat detection in the oral cavity has been thought to primarily rely on textural and olfactory cues; however recent research has fuelled speculation on a further role for the gustatory system and the existence of a specific taste for fatty acids (Mattes, 2009). The CD36 gene has been implicated in a number of functions, including facilitating the transport of long chain fatty acids into muscle and adipose tissue (Hajri et al., 2007; C. Martin et al., 2011; Silverstein & Febbraio, 2009). Recently, evidence is accumulating for the role of CD36 as a potential oral taste receptor for fatty acids (Laugerette et al., 2005; Simons & Boon, 2011; Takeda, Sawano, Imaizumi, & Fushiki, 2001).

Human research examining allelic variation in the CD36 gene has implicated several common polymorphisms that are associated with oral fatty acid detection thresholds (Pepino, Love-Gregory, Klein, & Abumrad, 2012) and the perception of fat content and fat preference (Keller et al., 2012). Pepino et al. (2012) investigated whether the A-allele in the rs1761667 SNP was associated with oral sensitivity to fat in twenty-one obese adults. They found that individuals who were G-allele homozygous showed higher oral fat sensitivity than those who were A-allele homozygous whereas heterozygotes' detection thresholds were intermediate (Pepino et al., 2012). Keller et

al. (2012) investigated five CD36 polymorphisms in a sample of healthy African American adults. Individuals with the rs1761667 AA genotype reported a greater perception of creaminess of salad dressings compared to those with the GG or GA genotype, independent of their actual fat content. The authors proposed that these individuals might be less sensitive in discriminating between the different salad dressings. Importantly, the individuals with the AA genotype reported a higher liking for added fats and oils (defined as butter, cooking oil etc). The implication of these early findings is that increased sensitivity to fat may influence intake of fat in the diet with potential consequences for body weight. Research in large cohorts has provided mixed evidence for associations between CD36 and measures of BMI and abdominal fat (Gertow et al., 2004). Bokor et al. (2010) investigated the relationship between four CD36 SNPs and percentage body fat and BMI in 1,151 adolescents and reported that having the major allele of these SNPs was associated with decreased BMI and percentage body fat (Bokor et al., 2010). However, other research regarding CD36 and BMI has been less consistent with some research supporting an association (Heni et al., 2010) and some not (Choquet et al., 2010). These findings suggest that variation in CD36 genotype may be associated with the intake of and liking for high-fat foods. Furthermore, increased sensitivity to fat taste may have implications for body composition and body weight perhaps via a potential resistance to dietary fat-induced weight gain.

### **9.2.3 Obesity related genes**

Genome-wide association studies have identified SNPs in two genes, FTO and MC4R, which have been consistently associated with increased body weight and enhanced risk for obesity (Frayling et al., 2007; Loos et al., 2008). Frayling et al. (2007) identified that the A allele of rs9939609 in the FTO gene was associated with an increase in BMI and greater prevalence of type 2 diabetes, with each copy of the A allele additively increasing the risk for obesity. This association has since been replicated in several independent cohorts (Hakanen et al., 2009; Hunt et al., 2012; Villalobos-Comparán et al., 2008). Further examination of the FTO gene has

suggested that its effect on body weight may be mediated by increased intake of palatable foods and decreased responsiveness to satiety signals rather than an action on energy expenditure (Cecil, Tavendale, Watt, Hetherington, & Palmer, 2008; Haupt et al., 2008; Wardle et al., 2008). The functional significance of FTO is not currently understood, however recent evidence has emerged that FTO may have a functional role in cellular nutrient sensing, which may help to explain the impact of polymorphisms in FTO on food preference and energy intake (Gulati et al., 2013).

Rare mutations in the MC4R gene are associated with early onset monogenic obesity (Farooqi & O'Rahilly, 2005). More common variations have been associated with an increase in body weight and a greater tendency to engage in binge eating behaviour (Branson et al., 2003). Loos et al. (2008) found that variation at rs17782313 in the MC4R gene was associated with a higher BMI in adults and children. Further research has replicated and extended this finding by showing that the C allele of rs17782313 is associated with increased waist circumference and fat mass across several different cohorts (Beckers et al., 2011; Cheung et al., 2010; Grant et al., 2009). Research examining a phenotype for MC4R variants has found that the C allele of rs17782313 is associated with increased energy intake, increased levels of dietary fat and enhanced risk of weight gain and diabetes in a large sample of females studied over 10 years (Qi, Kraft, Hunter, & Hu, 2008). Further evidence has supported the association between MC4R and increased energy intake (Stutzmann et al., 2009). Therefore, based on previous research it was hypothesised that common variants in the FTO and MC4R gene will be associated with BMI, body composition and increased intake of palatable snack foods.

#### **9.2.4 Study aims**

The primary aim of this study was to assess whether there is a common profile of genetic markers for the binge eating phenotype, as identified and characterised in previous chapters. The secondary aim was to examine the association of theoretically

relevant common gene variants with eating behaviour, body composition and psychometric traits implicated in trait binge eating (intermediary phenotypes).

### **9.3 Method**

#### **9.3.1 Participants**

One hundred and eighty participants were recruited across two sites in the United Kingdom (University of Leeds and University of St. Andrews) following identical protocols and procedures. Among them, 26% were male, 37% were overweight or obese and 83% were Caucasian. Participants were selected from an initial screening process that firstly excluded those who were currently taking medication, currently dieting, reported a history of eating disorders, or were unfamiliar with or disliked the study foods. Forty-one participants from the previous studies in this thesis, who had completed the ad libitum eating task and the LFPQ, provided a saliva sample for genotyping for inclusion in the current analysis but did not undergo any new behavioural measures. All participants received £5 for their participation. The relevant institutional ethical review boards approved all procedures in the present study. Informed written consent was obtained prior to the study.

#### **9.3.2 Design**

The study was cross-sectional in design, with participants attending the laboratory on two occasions: for the first visit, participants fasted overnight and arrived at the research unit in the morning to undergo a measure of body composition and a provide a saliva sample for the processing of DNA; for the second visit, participants were required to fast for at least 3.5 hours before arriving at the research unit, where ad libitum snack intake, food hedonics and psychometric traits were assessed. Ad libitum snack intake and food hedonics were assessed while participants were in a fasted state.

### **9.3.3 Measures**

#### **9.3.3.1 Subjective appetite sensations**

Measures of hunger, fullness, desire to eat and prospective consumption were taken using 100-mm VAS and are described in greater detail in Chapter 5.

#### **9.3.3.2 Food hedonics: explicit liking, explicit wanting and implicit wanting**

Measures of explicit liking, explicit wanting and implicit wanting were assessed using the LFPQ, which is described in greater detail in Chapter 4.

#### **9.3.3.3 Psychometric questionnaires**

The Binge Eating Scale (BES; Gormally et al., 1982) and the Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) were administered to participants at the end of the study procedures in order to assess trait binge eating, and levels of restraint, disinhibition and hunger. To assess trait reward sensitivity, the three subscales of the Behavioural Activation subscale (BAS; Carver & White, 1994) were used.

#### **9.3.3.4 Energy intake**

##### **9.3.3.4.1 Ad libitum eating task**

To provide a measure of food choice and energy intake the ad libitum eating task presented in Chapter 7 was used in the current study.

#### **9.3.3.5 Anthropometrics and body composition**

Standing height without shoes was measured to the nearest 0.5cm using a stadiometer. Body weight was measured using an electronic balance and recorded to the nearest 0.1kg. Waist circumference (cm) was measured 1cm above the top of the participants' naval after expiration. Bioelectrical impedance (model BC418MA; Tanita Europe B.V., UK) was used at both sites and in addition, air plethysmography (Bodpod, Concord, CA, USA) was used at the Leeds site in order to obtain an

estimate of participants' fat mass, fat free mass and percentage body fat. Measures of anthropometrics and body composition were taken following an overnight fast.

#### **9.3.3.6 Genotyping**

Participants were genotyped for seventeen single nucleotide polymorphisms (SNPs) across thirteen genes (see Table 9.1 for genes and SNP information) using the Sequenom MassArray system (Sequenom, Inc) and Taqman based approach. A saliva sample of approximately 2ml total volume was collected following manufacturer's instructions from each participant using Oragene DNA kits (#OG-250, DNA Genotek Inc, Ontario, Canada). In order to assure quality control during genotyping 5% of participants provided a second saliva sample so that they could be re-genotyped. The PCR cycling conditions involved 94°C for 30 seconds followed by 94°C for 5 seconds and 5 cycles of 52°C for 5 seconds and 80°C for 5 seconds for a total of 40 cycles, and then 72°C for 3 minutes and 4°C forever. Genotyping was performed at the Biomedical Research Centre at Ninewells Hospital and Medical School, University of Dundee, UK.



Table 9.1 Function of the identified reward, taste, and obesity related genes and the SNPs within them selected for analysis

	Gene	Gene function	SNP	SNP variants	Frequency <sup>1</sup>
<b>Reward related</b>	OPRM1 (opioid receptor mu 1)	Encodes the mu opioid receptor	rs1799971	A>G	0.19
			rs495491	T>C	0.31
	DRD2 (dopamine receptor D2)	Encodes the D2 subtype of receptor	rs6277	C>T	0.27
	ANKK1 (ankyrin repeat and kinase domain containing 1)	Linked to DRD2 receptors	rs1800497	C>T	0.30
<b>Taste related</b>	CD36 (cluster of differentiation 36)	Putative fat taste receptor.	rs2151916	T>C	0.35
			rs1761667	G>A	0.43
	TAS1R2 (taste receptor type 1 member 2)	Encodes sweet taste receptor T1R2	rs35874116	T>C	0.27
	TAS1R3 (taste receptor type 1 member 3)	Encodes human homolog of mouse Sac – sweet taste receptor	rs307355	C>T	0.22
			rs35744813	G>A	0.25
	TAS2R38 (taste receptor type 2 member 38)	Encodes a receptor for bitterness perception	rs1726866	C>T	0.41
<b>Obesity related</b>	SLC2A2 (solute carrier family 2 member 2)	Encodes a glucose sensor/transporter	rs5400	C>T	0.19
	FTO ( <i>fat mass and obesity associated gene</i> )	Possible role in control of food intake and choice	rs9939609	T>A	0.36
			rs1121980	C>T	0.37
	MC4R ( <i>melanocortin 4 receptor</i> )	Encodes for a melanocortin receptor	rs17782313	T>C	0.22

Note: <sup>1</sup>Frequency of the minor allele was taken from <http://www.ncbi.nlm.nih.gov/snp> using the 1000 Genome population.

### **9.3.4 Procedure**

Participants attended the laboratory for one morning visit following an overnight fast and one lunch time visit following a 3.5 hours fast. During the morning visit participants height, weight, waist circumference and body composition were measured. Additionally, during this session participants provided saliva samples for processing of DNA. During the lunchtime visit participants completed the LFPQ, followed by the ad libitum eating task. Participants completed ratings of subjective appetite at the beginning of this test session, and after each event in the procedure. At the end of the study procedures participants completed the psychometric questionnaires followed by written and verbal debriefing. Finally, they were compensated for their time before leaving the study.

### **9.3.5 Data Analysis**

Repeated measures ANOVAs were used to examine the influence of trait binge eating on appetite sensations, energy intake and food hedonic variables. Independent t-tests were used to examine the differences between the high and low binge eating groups. Chi-square analyses were used to determine whether there was a greater frequency of genotype underlying high or low scores on trait binge eating and to assess whether each gene was within Hardy-Weinberg equilibrium. For each SNP, data were analysed by entering genotype as a between-subjects variable with either three levels (e.g. CC vs. CT vs. TT) or two levels (e.g. T+ vs. T-). To examine the effect of genotype on body composition, anthropometric measures and snack food intake, analysis of covariance were used. The following covariates were used: for snack food intake, fat mass, percentage body fat and waist circumference – gender, age and site were controlled for; for BMI – age and site was controlled for; for fat free mass – gender was controlled for; finally, for food hedonics – site was controlled for. Covariates were identified using Pearson's correlations and independent t-tests. DNA for fifteen participants did not amplify during PCR and therefore they were excluded from the analysis. Furthermore, the genotyping of TAS1R3 was unsuccessful and so was not included in the analysis. Where

appropriate, Greenhouse-Geisser probability levels were used to adjust for non-sphericity. Post hoc analyses were conducted on significant interactions using the Bonferroni correction. An  $\alpha$ -level of 0.05 was used to determine statistical significance.

## 9.4 Results I

### 9.4.1 Sample characteristics according to site of collection

Sample characteristics of age, BMI, body composition and psychometric traits are shown in Table 9.2 for the overall sample, and the sample according to site of collection. Results from independent samples t tests showed that individuals in the Leeds sample had a higher BMI [ $t(178) = 2.87, p < 0.01$ ], waist circumference [ $t(176) = 5.59, p < 0.01$ ], fat mass [ $t(166) = 3.10, p < 0.01$ ], percentage body fat [ $t(166) = 3.01, p < 0.01$ ], disinhibition score [ $t(176) = 5.59, p < 0.01$ ] and trait binge eating score [ $t(173) = 2.16, p < 0.03$ ] compared with individuals from the St. Andrews sample.

Table 9.2 Mean (standard deviation) age, anthropometrics, body composition and psychometric trait characteristics for the overall sample and the two collection sites.

	Overall	Leeds	St. Andrews
Gender (F:M)	133:47	71:24	62:23
Age	26.04 (9.01)	26.74 (9.46)	25.26 (8.47)
BMI (kg/m <sup>2</sup> )	23.51 (3.84)	24.28 (4.24)**	22.66 (3.14)**
Fat mass (kg)	17.69 (8.53)	19.71 (9.03)**	15.74 (7.56)**
Body fat (%)	25.52 (8.89)	27.55 (8.78)**	23.52 (8.58)**
Lean mass (kg)	50.13 (9.53)	50.09 (10.05)	50.16 (9.06)
Waist (cm)	80.02 (11.23)	84.18 (12.43)**	75.47 (7.51)**
Restraint	8.32 (4.83)	8.74 (4.85)	7.79 (4.78)
Disinhibition	6.90 (3.60)	7.56 (3.76)**	6.12 (3.26)**
Hunger	6.06 (3.16)	6.47 (3.36)	5.57 (2.84)
Binge eating score	9.84 (6.92)	10.86 (7.36)*	8.63 (6.18)*

*Note:* Body composition assessed using bioelectrical impedance analysis.

\* $p < 0.05$ ; \*\* $p < 0.01$

#### 9.4.2 Effect of trait binge eating on food intake and appetite variables

To assess the effect of trait binge eating on food intake and appetite variables, the sample was divided into high and low trait binge eating groups using a stratified tertile split. Males and females were grouped separately; for males, individuals scoring  $\geq 10$  were categorised as high scorers and individuals scoring  $\leq 5$  were categorised as low scorers; for females, individuals scoring  $\geq 13$  were categorised as high scorers and individuals scoring  $\leq 6$  were categorised as low scorers. The high and low scoring males and females were then combined to create the high and low scoring binge eating groups. Table 9.3 summarises the characteristics of the newly created groups.

Table 9.3 Mean (standard deviation) age, anthropometrics, body composition and psychometric trait characteristics for the stratified tertile split of individuals scoring high and low in trait binge eating.

	Low scorers	High scorers
Gender (F:M)	47:12	47:15
Age	28.27 (10.26)	25.65 (8.58)
BMI (kg/m <sup>2</sup> )	22.09 (2.47)***	25.18 (4.64)***
Fat mass (kg)	15.25 (6.53)**	20.87 (9.87)**
Body fat (%)	23.73 (7.80)***	28.45 (9.14)***
Lean mass (kg)	48.43 (8.78)	50.43 (10.43)
Waist (cm)	75.31 (7.85)***	84.98 (13.16)***
Restraint	7.68 (4.58)	9.54 (5.10)
Disinhibition	3.90 (2.65)***	9.84 (2.86)***
Hunger	4.45 (2.46)***	7.61 (3.41)***
Binge eating score	3.31 (1.79)***	17.21 (5.70)***

*Note:* Body composition assessed using bioelectrical impedance analysis.

\*\* $p < 0.01$ ; \*\*\* $p < 0.001$

As expected, the high scorers had a significantly higher trait binge eating score compared to low scorers [ $t(119) = 17.93, p < 0.001$ ]. High scorers also scored higher in trait disinhibition [ $t(119) = 11.79, p < 0.001$ ] and hunger [ $t(119) = 5.71, p < 0.001$ ] compared to low scorers. When anthropometrics and body composition were analysed, it was revealed that high scorers had a significantly higher BMI [ $t(119) = 4.53, p < 0.001$ ], waist circumference [ $t(119) = 4.84, p < 0.001$ ], fat mass [ $t(108) = 3.48, p < 0.01$ ] and percentage body fat [ $t(108) = 2.89, p < 0.01$ ] compared to low scorers, while there were no difference in levels of lean mass [ $t(108) = 1.08, p > 0.05$ ].

#### **9.4.2.1 Subjective appetite sensations**

There was a main effect of time on ratings of hunger, fullness, prospective consumption and desire to eat [ $F(2, 234) = 102.43, p < 0.001$ ;  $F(2, 234) = 99.21, p < 0.001$ ;  $F(2, 234) = 76.15, p < 0.001$ ;  $F(2, 234) = 57.87, p < 0.001$ , respectively]. When these effects were examined it was revealed that participants' ratings of hunger, prospective consumption and desire to eat significantly increased following completion of the LFPQ [ $p < 0.001$ ] and then significantly decreased following the ad libitum eating task [ $p < 0.001$ ]. In addition, participants' ratings of fullness significantly decreased following the ad libitum eating task [ $p < 0.001$ ]. There was no effect of binge eating group on ratings of hunger, fullness, prospective consumption or desire to eat [ $F(2, 234) = .251, p > 0.05$ ;  $F(2, 234) = .705, p > 0.05$ ;  $F(2, 234) = .306, p > 0.05$ ;  $F(2, 234) = .472, p > 0.05$ , respectively].

#### **9.4.2.2 Food choice and intake**

##### **9.4.2.2.1 Ad libitum eating task**

Table 9.4 shows that high scorers consumed more energy from the ad libitum snack task compared to low scorers [ $F(1, 117) = 5.87, p < 0.02$ ]. However, when estimated energy requirements were taken into consideration (by adjusting for RMR) there were no differences between the groups in energy consumption [ $F(1, 117) = 1.61,$

$p>0.05$ ]. There was a non-significant trend for high scorers to consume more energy from sweet foods [ $F(1, 117) = 2.78, p=0.08$ ].

Table 9.4 Mean (standard deviation) energy intake from the ad libitum eating task for high and low scorers

	Low scorers (n=62)	High scorers (n=59)
Overall energy intake	370.18 (184.24)*	460.90 (222.13)*
Sweet energy intake	197.68 (133.76)	267.38 (156.58)
Savoury energy intake	172.49 (106.93)	193.52 (114.70)
EI:EEE snacks	.26 (.13)	.31 (.16)

*Abbreviations:* EI energy intake; EEE estimated energy expenditure

\* $p<0.05$

#### 9.4.2.2.1.1 Effect of gender on food choice and energy intake

To consider the influence that gender and trait binge eating may have on food choice and energy intake, the outcome of the ad libitum eating task was analysed for male and female high and low scorers separately. Table 9.5 shows that female high scorers consumed more overall energy in the ad libitum eating task compared to female low scorers [ $F(1, 87) = 7.14, p<0.01$ ] and more energy from sweet foods [ $F(1, 87) = 6.46, p<0.01$ ]. There were no differences between the male groups with regards to overall energy intake [ $F(1, 22) = .306, p>0.05$ ] or intake of sweet foods [ $F(1, 22) = .359, p>0.05$ ]. When estimated energy requirements were taken into consideration the differences in energy intake remained significantly higher for female high scorers compared to low scorers [ $F(1, 87) = 4.71, p<0.02$ ].

Table 9.5 Mean (standard deviation) energy intake from the ad libitum snack task for male and female high and low scorers

	Females		Males	
	Low (n=47)	High (n=47)	Low (n=12)	High (n=15)
Overall energy intake	333.18 (162.56)**	453.38 (188.74)**	515.05 (199.31)	483.52 (308.61)
Sweet energy intake	177.50 (118.63)**	264.26 (138.98)**	276.72 (164.12)	276.74 (206.13)
Savoury energy intake	155.68 (102.66)	189.10 (106.50)	238.33 (101.41)	206.77 (139.82)
EI:EEE snacks	.244 (.118)*	.309 (.136)*	.334 (.138)	.293 (.217)

*Abbreviations:* EI energy intake; EEE estimated energy expenditure

\* $p < 0.05$ ; \*\* $p < 0.01$

### 9.4.3 Effect of trait binge eating on food hedonics

#### 9.4.3.1 Explicit liking

An interaction between taste and group [ $F(1, 116) = 7.23, p < 0.01$ ] revealed that low scorers had lower explicit liking for sweet foods compared to high scorers. Post hoc analysis revealed the interaction was driven by lower explicit liking for high-fat sweet foods (see Table 9.6).

Table 9.6 Explicit liking ratings (mm) for high and low scorers

	Low scorers (n=62)	High scorers (n=59)
HFSA	58.21 (17.89)	54.88 (23.09)
LFSA	57.89 (18.82)	53.77 (19.17)
HFSW	46.09 (24.95)*	55.08 (24.10)*
LFSW	50.37 (15.08)	54.26 (19.40)

\* $p < 0.05$

### 9.4.3.2 Explicit wanting

An interaction between taste and group [ $F(1, 116) = 8.60, p < 0.01$ ] revealed that low scorers had lower explicit wanting for sweet foods compared to high scorers. Post hoc analysis revealed that the interaction was driven by lower explicit wanting for high-fat sweet foods (see Table 9.7).

Table 9.7 Explicit wanting ratings (mm) for high and low scorers

	Low scorers (n=62)	High scorers (n=59)
HFSA	56.54 (19.90)	53.58 (22.35)
LFSA	56.46 (18.57)	52.12 (19.09)
HFSW	42.34 (25.91)*	53.28 (24.65)*
LFSW	47.96 (16.64)	52.47 (18.25)

\* $p < 0.05$

### 9.4.3.3 Implicit wanting

An interaction between taste and group [ $F(1, 114) = 5.49, p < 0.02$ ] revealed that low scorers had a greater implicit wanting for savoury foods compared to high scorers. Additionally, an interaction between taste, fat and group revealed that high scorers had lower implicit wanting for high fat savoury foods [ $p < 0.02$ ] and greater implicit wanting for high fat sweet foods [ $p < 0.01$ ] compared to low scorers (see Table 9.8).

Table 9.8 Implicit wanting ratings (D-RT) for high and low scorers

	Low scorers (n=62)	High scorers (n=59)
HFSA	.144 (.389)*	-.071 (.550)*
LFSA	.170 (.459)	.128 (.497)
HFSW	-.342 (.492)**	.094 (.450)**
LFSW	.092 (.471)	.073 (.422)

\* $p < 0.05$ ; \*\* $p < 0.01$



#### 9.4.4 Genotype analyses: is there a common genotype underlying trait binge eating?

Chi-square analyses were conducted to test genotype frequency differences between individuals scoring high or low in trait binge eating. Due to the rare occurrence of the A1/A1 genotype for ANKK1 rs1800497 this group were combined with the A1/A2 genotype to form the A1+ group, whereas individuals with the A2/A2 genotype formed the A1- group. Similarly, due to low frequencies for the minor allele, participants were defined as G+ or G- for OPRM1 rs1799971, C+ or C- for OPRM1 rs495491, T+ or T- for SLC2A2 rs5400, C+ or C- for TAS1R2 rs35874116, and C+ and C- for MC4R rs17782313.

##### 9.4.4.1 Reward related genes

The results showed that there were no differences between high and low scorers in genotype frequency for any of the reward related genes (see Table 9.9).

Table 9.9 2x2 and 2x3 contingency tables for comparing reward related genotypes in individuals scoring high and low in trait binge eating.

OPRM1	rs1799971	G+	G-	Total	
	High scorers	14 (24.1%)	44 (75.9%)	58	
	Low scorers	21 (35.6%)	38 (64.4%)	59	
	Total	35	82	117	
	$\chi^2 = 1.83$ ; p= .176				
	rs495491	C+	C-	Total	
	High scorers	25 (43.1%)	33 (56.9%)	54	
	Low scorers	26 (48.1%)	28 (51.9%)	58	
	Total	51	61	112	
	$\chi^2 = .287$ ; p= .592				
DRD2	rs6277	CC	CT	TT	Total
	High scorers	20 (34.5%)	28 (48.3%)	10 (17.2%)	58
	Low scorers	12 (23.1%)	25 (48.1%)	15 (28.8%)	52
	Total	32	53	25	110
	$\chi^2 = 2.85$ ; p= .240				
ANKK1	rs1800497	A1+	A1-	Total	
	High scorers	30 (52.6%)	27 (47.4%)	57	
	Low scorers	22 (41.5%)	31 (58.5%)	53	
	Total	52	58	110	
	$\chi^2 = 1.36$ ; p= .243				

#### 9.4.4.2 Taste related genes

Chi-square analyses revealed that there were no differences between high and low scorers in genotype frequency for any of the taste related genes (see Table 9.10).

Table 9.10 2x2 and 2x3 contingency tables for comparing taste related genotypes in individuals scoring high and low in trait binge eating.

CD36	rs2151916	TT	TC	CC	Total
	High scorers	23 (39.7%)	28 (48.3%)	7 (12.1%)	58
	Low scorers	16 (29.6%)	26 (48.1%)	12 (22.2%)	54
	Total	39	54	19	112
	$\chi^2 = 2.51; p = .286$				
	rs1761667	GG	GA	AA	Total
	High scorers	19 (32.8%)	28 (48.3%)	11 (19.0%)	58
	Low scorers	12 (22.6%)	28 (52.8%)	13 (24.5%)	53
	Total	31	56	24	111
	$\chi^2 = 1.53; p = .466$				
TAS1R2	rs35874116	C+	C-		Total
	High scorers	18 (30.5%)	41 (69.5%)		59
	Low scorers	18 (33.3%)	36 (66.7%)		54
	Total	36	77		113
	$\chi^2 = .104; p = .748$				
TAS2R38	rs1726866	CC	CT	TT	Total
	High scorers	9 (17.6%)	24 (47.1%)	18 (35.3%)	51
	Low scorers	12 (25.0%)	15 (31.2%)	21 (43.8%)	48
	Total	21	39	39	99
	$\chi^2 = 2.65; p = .266$				
SLC2A2	rs5400	T+	T-		Total
	High scorers	15 (26.3%)	42 (73.7%)		57
	Low scorers	17 (30.9%)	38 (69.1%)		55
	Total	32	80		112
	$\chi^2 = .289; p = .591$				

#### 9.4.4.3 Obesity related genes

Chi-square analyses revealed that there were no differences between high and low scorers in genotype frequency for any of the obesity related genes (see Table 9.11).

Table 9.11 2x2 and 2x3 contingency tables for comparing obesity related genotypes in individuals scoring high and low in trait binge eating.

FTO	rs9939609	TT	TA	AA	Total
	High scorers	21 (36.8%)	24 (42.1%)	12 (21.1%)	57
	Low scorers	18 (35.3%)	25 (49.0%)	8 (15.7%)	51
	Total	39	49	20	108
	$\chi^2 = .720$ ; p= .698				
	rs1121980	CC	CT	TT	Total
	High scorers	20 (34.5%)	27 (46.6%)	11 (19.0%)	58
	Low scorers	18 (34.0%)	26 (49.1%)	9 (17.0%)	53
	Total	38	53	20	111
	$\chi^2 = .099$ ; p= .952				
MC4R	rs17782313	C+	C-		Total
	High scorers	35 (60.3%)	23 (39.7%)		58
	Low scorers	33 (61.1%)	21 (38.9%)		54
	Total	68	44		112
	$\chi^2 = .007$ ; p= .934				

## 9.5 Discussion I

The primary aim of the current study was to examine whether a common underlying genotype could be identified for the trait binge eating behavioural phenotype that has been characterised in the previous chapters of this thesis. Using a candidate gene approach, genes of interest were identified from a literature search to find SNPs that have previously been studied in relation to binge eating, preferences for foods high in sugar or fat, and obesity.

Higher trait binge eating scores were associated with a larger waist circumference, and greater amounts of fat mass and percentage body fat however this finding appeared to be driven by the greater number of overweight or obese individuals in the high scoring group. In line with this, it was also shown that high scorers had a higher BMI compared to low scorers. In the previous chapters in this thesis, the trait

binge eating groups have been formed matching for age and BMI to overcome this issue. Therefore future research should stratify the sample based on both gender and BMI category in order to assess the influence of trait binge eating on anthropometric and body composition variables.

In accordance with our previous findings, there was a trend for high scorers to exhibit a preference for high-fat sweet foods compared to low scorers, although this did not reach statistical significance. In addition, there were no differences in total energy consumed from the ad libitum eating task. However, when gender was taken into consideration, female high scorers consumed more energy from the ad libitum eating task, and exhibited a greater preference for sweet foods compared to female low scorers. The same pattern of intake was not observed in males. To our knowledge, gender differences in the preferred foods of individuals with the tendency to binge eat has not been examined before. However, previous research does suggest that while there are similarities between male and female binge eaters there are also some key gender differences (Barry, Grilo, & Masheb, 2002; Mitchell & Mazzeo, 2004; Striegel-Moore et al., 2009; Tanofsky, Wilfley, Spurrell, Welch, & Brownell, 1997). For example, Tanofsky et al. (1997) examined levels of eating psychopathology and psychological functioning in individuals with BED. They found that females reported experiencing greater levels of emotional eating whereas males reported experiencing greater psychiatric symptomatology, including greater reports of substance abuse. Interestingly, Wansink, Cheney, and Chan (2003) found that comfort foods differed between males and females; males preferred warm, meal-related comfort foods such as casseroles or steak whereas females preferred more snack related comfort foods such as chocolate and ice cream. Furthermore, in a large survey based study, Striegel-Moore et al. (2009) reported that females were more likely than males to endorse experiencing a loss of control over eating, a feeling that is key in binge eating behaviour. Therefore, it can be tentatively suggested that the

tendency to binge eat in males may be quantitatively and qualitatively different to the tendency to binge eat in females.

In contrast with the previous chapters in this thesis, the current study did not find that greater trait binge eating scores were associated with enhanced liking for high-fat sweet foods. However it was demonstrated that high scorers had greater implicit wanting for high-fat sweet foods (although to a lesser degree than in the previous chapters) compared to low scorers. One possible reason for the disparity in findings may be due to small procedural or sample differences between sites that could have accumulated sufficient noise (error variance) in the study to mask some reliable effects. For example, at the Leeds site, participants completed the LFPQ alone in an experimental cubicle while at the St. Andrews site, some participants would have completed the LFPQ in the presence of others which may have implications for the results with regards to reduced attention during the completion of the implicit wanting trials and social desirability effects when rating the foods. Further to this, the two populations recruited appeared to be fairly distinct which would have increased the variability within the high and low scoring groups. The current study did not use a matched pairs design in which individuals were recruited on the basis of their binge eating score and then age and BMI matched to either the binge type group or the non-binge group. In future research, employing this more intensive selection procedure would help to avoid a disproportionate number of overweight or obese individuals in the high scoring group.

Finally, the frequency of allelic variants did not differ between high or low scorers suggesting that, with regards to the SNPs examined, there was no common underlying genotype for the behavioural binge eating phenotype. The secondary aim of the current study was to examine the association of the identified theoretically relevant common gene variants with eating behaviour, body composition and psychometric traits that appear to characterise trait binge eating (intermediary phenotypes).

## 9.6 Results II

### 9.6.1 Reward related genes

#### 9.6.1.1 Genotype frequencies

Genotype frequency analyses for the reward related genes are reported in Table 9.12. The results indicated that genotype frequency for OPRM1 rs1799971 and rs495491 did not deviate from Hardy-Weinberg Equilibrium [ $\chi^2 = 0.31$ ,  $p=0.58$ ;  $\chi^2 = 0.02$ ,  $p=0.89$ , respectively]. In addition, genotype frequency for DRD2 rs6277 and ANKK1 rs1800497 did not deviate from Hardy-Weinberg Equilibrium [ $\chi^2 = 1.39$ ,  $p=0.24$ ;  $\chi^2 = 1.09$ ,  $p=0.29$ , respectively]. An overall summary of the findings for this section can be found in Table 9.13.

Table 9.12 Genotype frequencies for OPRM1 rs1799971 and rs495491, DRD2 rs6277 and ANKK1 rs1800497

		No. of participants	Gender (F:M)	Genotype frequency %
<b>OPRM1</b>				
rs1799971	AA	121	93:28	68.8
	AG	51	36:15	29.0
	GG	4	0:4	2.3
<i>Frequency of G allele</i>				0.17
rs495491	TT	88	63:25	53.0
	TC	66	48:18	39.8
	CC	12	9:3	7.2
<i>Frequency of C allele</i>				0.27
<b>DRD2</b>				
rs6277	CC	46	26:20	27.9
	CT	76	61:15	46.1
	TT	43	32:11	26.1
<i>Frequency of T allele</i>				0.49
<b>ANKK1</b>				
rs1800497	A2/A2	91	71:20	54.8
	A2/A1	61	43:18	36.7
	A1/A1	14	6:8	8.4
<i>Frequency of A1 allele</i>				0.27

### **9.6.1.2 Effect of OPRM1 genotype**

#### **9.6.1.2.1 Energy intake**

There was no effect of OPRM1 rs1799971 or rs495491 on energy intake from the ad libitum eating task and there were no differences in consumption of sweet or savoury foods for rs1799971 or rs495491 (see Appendix 9 for outcome of the analysis).

#### **9.6.1.2.2 Anthropometrics and body composition**

There was no effect of OPRM1 rs1799971 or rs495491 on anthropometrics or body composition (see Appendix 9 for outcome of the analysis).

#### **9.6.1.2.3 Food hedonics**

There was no effect of OPRM1 rs1799971 or rs495491 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### **9.6.1.2.4 Psychometric traits**

There was no effect of OPRM1 rs1799971 or rs495491 on any of the measured psychometric traits (see Appendix 9 for outcome of the analysis).

### **9.6.1.3 Effect of DRD2 genotype**

#### **9.6.1.3.1 Energy intake**

There was an effect of DRD2 rs6277 on total energy intake from the ad libitum eating task [ $F(2, 157) = 4.16, p < 0.02$ ] and on energy intake from sweet snack foods [ $F(2, 157) = 5.73, p < 0.01$ ]. Figure 9.1 shows that individuals with the CT genotype consumed more energy overall and more energy from sweet foods compared to individuals with the CC genotype. There were no differences in energy intake from savoury foods [ $F(1, 157) = .372, p > 0.05$ ].

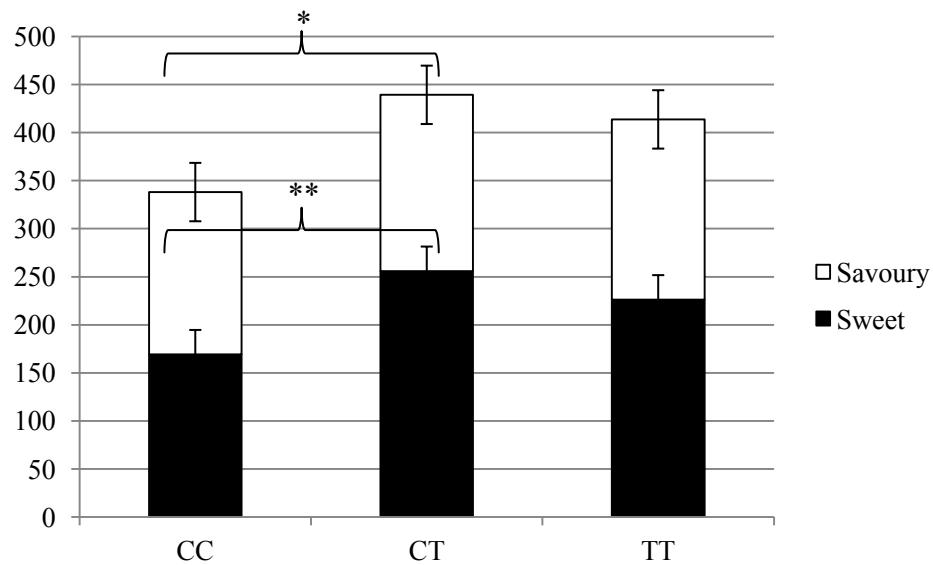


Figure 9.1 Energy intake (kcal) from the ad libitum intake task according to DRD2 rs6277 genotype

\* $p < 0.05$ ; \*\* $p < 0.01$

#### 9.6.1.3.2 Anthropometrics and body composition

There was an effect of DRD2 rs6277 on BMI [ $F(2, 160) = 5.04, p < 0.01$ ]. Post hoc analyses revealed that individuals with the CC genotype [M: 24.92, SE: .525] had a significantly higher BMI compared to those with the TT genotype [M: 22.68, SE: .539,  $p < 0.01$ ] and the CT genotype [M: 23.20, SE: .405,  $p < 0.03$ ]. Additionally, there was an effect of rs6277 on waist circumference [ $F(2, 160) = 3.34, p < 0.04$ ] with individuals with the CC genotype [M: 83.12, SE: 1.43] having a larger waist circumference compared to those with the TT genotype [M: 78.09, SE: 1.11,  $p < 0.05$ ] genotype. There were no differences in waist circumference between CC and CT [M: 79.33, SE: 1.11], or CT and TT. Finally, in the individuals who had their body composition assessed using air plethysmography, there was an effect of genotype on percentage body fat [ $F(2, 71) = 3.33, p < 0.05$ ]. Post hoc analyses revealed that those with the CC genotype [M: 32.10, SE: 1.57] had a higher percentage body fat compared to those with the CT genotype [M: 25.87, SE: 1.57]. There were no differences in percentage body fat between CC and TT [M: 27.60, SE: 2.11], or CT and TT.



#### **9.6.1.3.3 Food hedonics**

There was no effect of DRD2 rs6277 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### **9.6.1.3.4 Psychometric traits**

There was no effect of DRD2 rs6277 on any of the measured psychometric traits (see Appendix 9 for outcome of the analysis).

### **9.6.1.4 Effect of ANKK1 genotype**

#### **9.6.1.4.1 Energy intake**

There was no effect of ANKK1 rs1800497 on energy intake from the ad libitum eating task and there were no differences in consumption of sweet or savoury foods (see Appendix 9 for outcome of the analysis).

#### **9.6.1.4.2 Anthropometrics and body composition**

The presence of the A1 allele was associated with BMI [ $F(1, 162) = 4.41, p < 0.05$ ]; individuals in the A1+ group had a higher BMI [ $n = 75$ ; M: 24.18, SE: .420] compared to those in the A1- group [ $n = 91$ ; M: 22.98, SE: .381].

#### **9.6.1.4.3 Food hedonics**

There was no effect of ANKK1 rs1800497 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### **9.6.1.4.4 Psychometric traits**

There was an effect of ANKK1 rs1800497 on two of the BAS subscales. Individuals in the A1+ group [M: 12.36, SE: .328] had lower BAS fun-seeking scores [ $F(1, 160) = 5.25, p < 0.02$ ] compared to those in the A1- group [M: 15.37, SE: .292]. Additionally, individuals in the A1+ group [M: 19.35, SE: .110] had lower BAS

Reward Responsiveness scores [ $F(1, 160) = 5.15, p < 0.03$ ] compared to those in the A1- group [ $M: 22.39, SE: .891$ ].

Table 9.13 Reward related genes summary table

	OPRM1		DRD2	ANKK1
	rs1799971	rs495491	rs6277	rs1800497
<i>Energy intake</i>				
Ad libitum eating task			✓	
Sweet snacks			✓	
Savoury snacks				
<i>Body composition</i>				
BMI			✓	✓
Waist circumference			✓	
Fat mass				
% Body fat			✓	
Fat free mass				
<i>Food hedonics</i>				
Explicit liking				
Explicit wanting				
Implicit wanting				
<i>Psychometric traits</i>				
Restraint				
Disinhibition				
Hunger				
BAS				✓
BIS				
STR				
BES				

## 9.6.2 Taste related genes

### 9.6.2.1 Genotype frequencies

Genotype frequency analyses for the taste related genes are reported in Table 9.14. Genotype frequency for CD36 rs2151916 and rs1761667, TAS1R2 rs35874116 and SLC2A2 rs5400 did not deviate from Hardy-Weinberg Equilibrium [ $\chi^2 = 0.04, p = 0.83$ ;  $\chi^2 = 0.05, p = 0.95$ ;  $\chi^2 = 1.19, p = 0.27$ ;  $\chi^2 = 0.12, p = 0.73$ , respectively]. However, while genotype frequency for TAS2R38 rs1726866 did not deviate from Hardy-Weinberg Equilibrium [ $\chi^2 = 3.57, p = 0.06$ ] the frequency of the T allele in the study sample (55%) was higher than in the population (41%). An overall summary of the findings for this section can be found in Table 9.17.

Table 9.14 Genotype frequency for CD36 rs2151916 and rs1761667, TAS1R2 rs35874116, TAS2R38 rs1726866 and SLC2A2 rs5400

		No. of participants	Gender (F:M)	Genotype frequency %
<b>CD36</b>				
rs2151916	TT	57	44:13	34.5
	TC	81	59:22	49.1
	CC	27	18:9	16.4
<i>Frequency of C allele</i>				0.41
rs1761667	GG	45	30:15	27.3
	GA	81	56:25	49.1
	AA	39	34:5	23.6
<i>Frequency of A allele</i>				0.48
<b>TAS1R2</b>				
rs35874116	TT	73	50:23	43.5
	TC	80	61:19	47.6
	CC	15	11:4	8.9
<i>Frequency of C allele</i>				0.33
<b>TAS2R38</b>				
rs1726866	CC	35	28:7	23.5
	CT	63	41:22	42.3
	TT	51	40:11	34.2
<i>Frequency of T allele</i>				0.55
<b>SLC2A2</b>				
rs5400	CC	120	85:35	72.3
	CT	43	35:8	25.9
	TT	3	2:1	1.8
<i>Frequency of T allele</i>				0.15

### 9.6.2.2 Effect of CD36 genotype

#### 9.6.2.2.1 Energy intake

There was no effect of CD36 rs2151916 or rs1761667 on energy intake from the ad libitum eating task or on energy from sweet or savoury foods (see Appendix 9 for the outcome of the analysis).

#### **9.6.2.2.2 Anthropometrics and body composition**

Variation at rs2151916 was associated with waist circumference [ $F(2, 159) = 3.84$ ,  $p < 0.02$ ]; individuals with the CC genotype [M: 75.22, SE: 1.85] had a smaller waist circumference than individuals with the CT genotype [M: 80.63, SE: 1.05;  $p < 0.03$ ] and those with the TT genotype [M: 81.01, SE: 1.25;  $p < 0.03$ ].

The effect of CD36 genotype on the air plethysmography assessment of body fat and fat free mass in the Leeds sample revealed, for rs2151916, individuals with the CC genotype had a significantly smaller waist circumference [ $F(2, 78) = 3.47$ ,  $p < 0.05$ ] and a significantly lower body mass index [ $F(2, 84) = 3.27$ ,  $p < 0.04$ ] and fat mass [ $F(2, 78) = 5.44$ ,  $p < 0.01$ ] compared to individuals with the CT or TT genotype (see Table 9.15).

For rs1761667, individuals with the GG genotype had a significantly lower fat mass [ $F(2, 78) = 3.66$ ,  $p < 0.03$ ] and percentage body fat [ $F(2, 78) = 3.94$ ,  $p < 0.04$ ] compared to those with the GA genotype, while the difference between GG and AA approached significance [ $p = 0.08$ ]. These effects appeared specific to markers of adiposity, as there were no differences in fat free mass between the genotypes [rs2151916:  $F(2, 78) = .465$ ,  $p > 0.05$ ; rs1761667:  $F(2, 78) = 0.30$ ,  $p > 0.05$ ].

Table 9.15 Mean (SEM) anthropometrics and body composition for the Leeds sample according to CD36 rs2151916 and rs1766671

rs2151916	TT (n=30)	TC (n=37)	CC (n=18)
BMI	24.81 (.79) <sup>***</sup>	24.94 (.73) <sup>b*</sup>	21.92 (.87) <sup>***/b*</sup>
Waist (cm)	87.03 (2.11) <sup>***</sup>	85.56 (2.07) <sup>b*</sup>	77.76 (2.43) <sup>***/b*</sup>
Fat mass (kg)	21.94 (1.76) <sup>***</sup>	20.88 (1.91) <sup>b**</sup>	12.73 (1.76) <sup>***/b**</sup>
Body fat (%)	30.82 (1.59) <sup>***</sup>	29.91 (1.89) <sup>b**</sup>	20.62 (2.43) <sup>***/b**</sup>
Fat free mass (kg)	47.11 (1.67)	49.46 (1.71)	49.10 (2.63)
rs1766671	GG (n=28)	GA (n=39)	AA (n=18)
BMI	23.51 (.79)	25.02 (.69)	24.31 (.87)
Waist (cm)	82.52 (2.24)	85.57 (2.05)	85.12 (2.83)
Fat mass (kg)	15.87 (1.79) <sup>a*</sup>	22.52 (1.85) <sup>a*</sup>	20.71 (2.52)
Body fat (%)	23.50 (2.16) <sup>a*</sup>	30.43 (1.76) <sup>a*</sup>	30.96 (2.18)
Fat free mass (kg)	49.13 (1.93)	49.11 (1.68)	46.98 (2.27)

*Note:* Body composition assessed using air plethysmography

\* $p < 0.05$ ; \*\* $p < 0.01$

#### 9.6.2.2.3 Food hedonics

There was no effect of CD36 rs2151916 or rs1761667 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### 9.6.2.2.4 Psychometric traits

There was no effect of CD36 rs2151916 or rs1761667 on any of the measured psychometric traits (see Appendix 9 for outcome of analysis).

### 9.6.2.3 Effect of TAS1R2 genotype

#### 9.6.2.3.1 Energy intake

There was no effect of TAS1R2 rs35874116 on energy intake from the ad libitum eating task or on energy from sweet or savoury foods (see Appendix 9 for the outcome of the analysis).

#### 9.6.2.3.2 Anthropometrics and body composition

Variation in TAS1R2 was associated with BMI [ $F(1, 164) = 8.14, p < 0.01$ ], waist circumference [ $F(2, 160) = 3.76, p < 0.03$ ], fat mass [ $F(1, 155) = 6.13, p < 0.02$ ] and percentage body fat [ $F(1, 155) = 5.64, p < 0.02$ ]. Post hoc analyses revealed that individuals in the C- group had a higher BMI compared to those in the C+ group. Additionally, the C+ group had a lower fat mass, percentage body fat and waist circumference compared to those in the C- group (see Table 9.16).

Table 9.16 Mean (SEM) anthropometrics and body composition according to TAS1R2 rs35874116

	C- (n=73)	C+ (n=95)
BMI	24.37 (.42)**	22.77 (.37)**
Waist (cm)	81.91 (1.10)*	78.34 (.98)*
Fat mass (kg)	19.22 (.91)**	16.19 (.81)**
Body fat (%)	26.74 (.78)*	24.25 (.69)*
Fat free mass (kg)	50.75 (.62)	49.61 (.54)

*Note:* Body composition assessed using bioelectrical impedance analysis

\* $p < 0.05$ ; \*\* $p < 0.01$

#### 9.6.2.3.3 Food hedonics

There was no effect of TAS1R2 rs35874116 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### **9.6.2.3.4 Psychometric traits**

There was no effect of TAS1R2 rs35874116 on any of the measured psychometric traits (see Appendix 9 for outcome of analysis).

#### **9.6.2.4 Effect of TAS2R38 genotype**

##### **9.6.2.4.1 Energy intake**

There were no differences in overall energy consumed from the ad libitum eating task [ $F(2, 141) = .233, p > 0.05$ ]; however, individuals with the CC genotype [M: 216.02, SE: 18.01] consumed more energy from savoury snack foods [ $F(2, 141) = 3.17, p < 0.05$ ] compared to those with the TT genotype [M: 160.04, SE: 14.85;  $p < 0.05$ ]. There were no differences in savoury snack food consumption between CC and CT [M: 195.96, SE: 13.69] or between TT and CT.

##### **9.6.2.4.2 Anthropometrics and body composition**

There was no effect of TAS2R38 rs1726866 on anthropometrics or body composition (see Appendix 9 for outcome of analysis).

##### **9.6.2.4.3 Food hedonics**

There was no effect of TAS2R38 rs1726866 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

##### **9.6.2.4.4 Psychometric traits**

Variation at TAS2R38 rs1726866 was associated with TFEQ Restraint [ $F(2, 139) = 3.47, p < 0.03$ ]. Post hoc analyses revealed a trend for individuals with the CC genotype [M: 7.14, SE: .81] to have lower levels of restraint compared to heterozygotes [M: 9.49, SE: .62;  $p = 0.07$ ]. There were no differences in restraint between CC and TT [M: 7.54, SE: .68] or between CT and TT. There were no other effects of TAS2R38 rs1726866 on psychometric traits see (see Appendix 9 for outcome of analysis).

### **9.6.2.5 Effect of SLC2A2 genotype**

#### **9.6.2.5.1 Energy intake**

Individuals in the T- group [ $n = 120$ ; M: 192.42, SE: 9.58] consumed significantly more energy from savoury snack foods compared to those in the T+ group [ $n = 46$ ; M: 153.79, SE: 15.48;  $F(1, 157) = 4.49$ ,  $p < 0.04$ ].

#### **9.6.2.5.2 Anthropometrics and body composition**

There was no effect of SLC2A2 rs5400 on anthropometrics or body composition (see Appendix 9 for outcome of analysis).

#### **9.6.2.5.3 Food hedonics**

There was a trend for variation at SLC2A2 to be associated with ratings of explicit liking [ $F(1, 156) = 3.24$ ,  $p = 0.07$ ]; T+ individuals had higher liking for sweet foods [M: 55.69, SE: 2.14] compared to T- individuals [M: 52.35, SE: 1.34]. Explicit liking ratings for savoury foods were similar for T- and T+ [M: 54.16, SE: 1.27; M: 53.80, 2.02, respectively]. There was no effect of SLC2A2 rs5400 on explicit wanting or implicit wanting according to fat content [ $F(2, 156) = .205$ ,  $p > 0.05$ ;  $F(2, 153) = .040$ ,  $p > 0.05$ , respectively] or taste [ $F(2, 156) = 2.37$ ,  $p > 0.05$ ;  $F(2, 153) = .371$ ,  $p > 0.05$ ] of the food images.

#### **9.6.2.5.4 Psychometric traits**

There was no effect of SLC2A2 rs5400 on any of the measured psychometric traits (see Appendix 9 for outcome of analysis).



Table 9.17 Taste related genes summary table

	CD36		TAS1R2	TAS2R38	SLC2A2
	rs2151916	rs1761667	rs35874116	rs1726866	rs5400
<i>Energy intake</i>					
Ad libitum eating task					
Sweet snacks					
Savoury snacks				✓	✓
<i>Body composition</i>					
BMI	✓		✓		
Waist circumference	✓		✓		
Fat mass	✓	✓	✓		
% Body fat		✓			
Fat free mass					
<i>Food hedonics</i>					
Explicit liking					✓
Explicit wanting					
Implicit wanting					
<i>Psychometric traits</i>					
Restraint				✓	
Disinhibition					
Hunger					
BAS					
BIS					
STR					
BES					

### 9.6.3 Obesity related genes

#### 9.6.3.1 Genotype frequencies

Genotype frequency analyses for the obesity related genes are reported in Table 9.18. Genotype frequency for FTO rs9939609 and rs1121980 or MC4R rs17782313 did not deviate from Hardy-Weinberg Equilibrium [ $X^2 = 0.09$ ,  $p=0.76$ ;  $X^2 = 0.25$ ,  $p=0.61$ ;  $X^2 = 0.83$ ,  $p=0.36$ , respectively]. An overall summary of the findings for this section can be found in Table 9.19.

Table 9.18 Genotype frequency for FTO rs9939609 and rs1121980 and MC4R rs17782313

		No. of participants	Gender (F:M)	Genotype frequency %
<b>FTO</b>				
rs9939609	TT	58	43:15	36.0
	TA	76	56:20	47.2
	AA	27	20:7	16.8
<i>Frequency of A allele</i>				0.41
rs1121980	CC	54	37:17	32.5
	CT	85	64:21	51.2
	TT	27	20:7	16.3
<i>Frequency of T allele</i>				0.42
<b>MC4R</b>				
rs17782313	TT	99	75:24	59.6
	TC	60	40:20	36.1
	CC	7	6:1	3.9
<i>Frequency of C allele</i>				0.22

### 9.6.3.2 Effect of FTO genotype

#### 9.6.3.2.1 Energy intake

Variation at rs9939609 was associated with a trend towards greater energy intake from the ad libitum eating task [ $F(2, 151) = 2.80, p=0.06$ ]. Further analysis revealed that individuals with the TA genotype consumed more energy from sweet snack foods compared to those with the TT genotype [ $F(2, 151) = 5.77, p<0.01$ ] (see Figure 9.2).

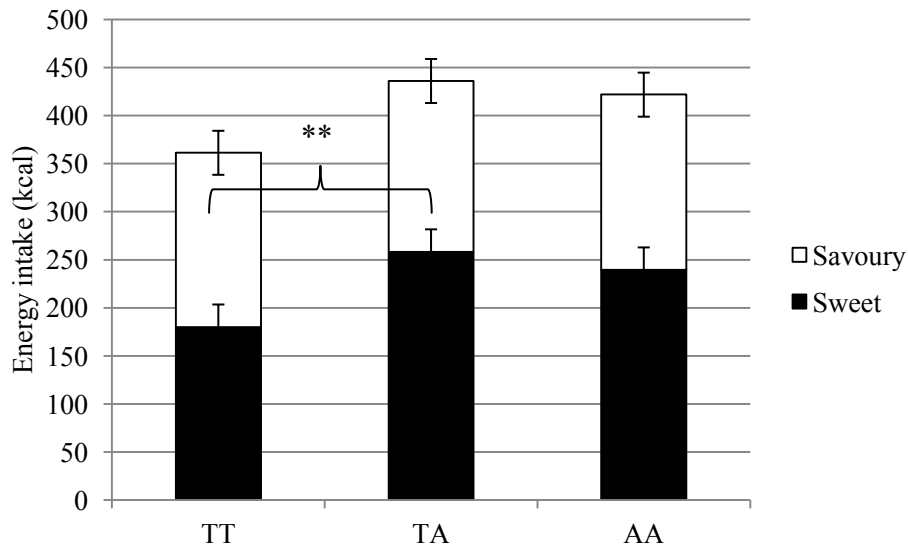


Figure 9.2 Energy intake (kcal) from the ad libitum eating task according to FTO rs9939609

**\*\* $p < 0.01$**

For rs1121980, there was an effect of genotype on total energy consumed from the ad libitum eating task [ $F(2, 156) = 3.54, p < 0.03$ ] and energy consumed from sweet snack foods [ $F(2, 156) = 5.66, p < 0.01$ ]. When these effects were explored, it was revealed that individuals with the CC genotype consumed less energy from the ad libitum eating task compared to CT individuals [ $p < 0.05$ ]. Additionally, the presence of the T allele appeared to be associated with greater consumption of sweet snack foods with both individuals with the CT [ $p < 0.01$ ] and TT [ $p < 0.05$ ] consuming more energy from sweet foods compared to individuals with the CC genotype (see Figure 9.3).

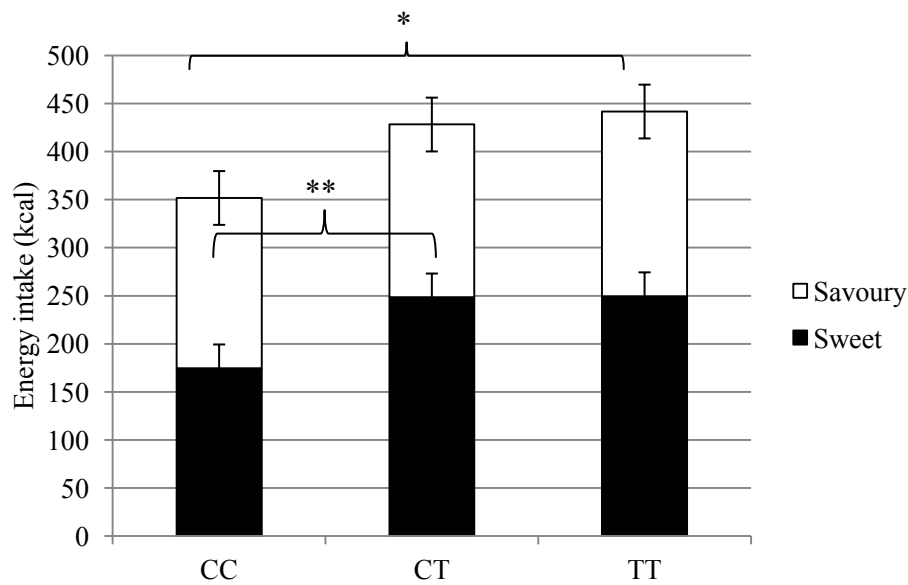


Figure 9.3 Energy intake (kcal) from the ad libitum eating task according to FTO rs1121980

\* $p<0.05$ ; \*\* $p<0.01$

#### 9.6.3.2.2 Anthropometrics and body composition

There was no effect of FTO rs9939609 or rs1121980 on anthropometrics or body composition (see Appendix 9 for outcome of analysis).

#### 9.6.3.2.3 Food hedonics

There was no effect of FTO rs9939609 or rs1121980 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### 9.6.3.2.4 Psychometric traits

There was no effect of FTO rs9939609 or rs1121980 on any of the measured psychometric traits (see Appendix 9 for outcome of analysis).

### **9.6.3.3 Effect of MC4R genotype**

#### **9.6.3.3.1 Energy intake**

There was no effect of MC4R rs17782313 on energy intake from the ad libitum eating task or on energy from sweet or savoury foods (see Appendix 9 for the outcome of the analysis).

#### **9.6.3.3.2 Anthropometrics and body composition**

There was no effect of MC4R rs17782313 on anthropometrics or body composition (see Appendix 9 for outcome of analysis).

#### **9.6.3.3.3 Food hedonics**

There was no effect of MC4R rs17782313 on explicit liking, explicit wanting or implicit wanting according to fat content or taste of the food images (see Appendix 9 for outcome of analysis).

#### **9.6.3.3.4 Psychometric traits**

There was no effect of MC4R rs17782313 on any of the measured psychometric traits (see Appendix 9 for outcome of analysis).

Table 9.19 Obesity related genes summary table

	FTO	MC4R
	rs9939609	rs1121980
	rs17782313	
<i>Energy intake</i>		
Ad libitum eating task	✓	✓
Sweet snacks	✓	✓
Savoury snacks		
<i>Body composition</i>		
BMI		
Waist circumference		
Fat mass		
% Body fat		
Fat free mass		
<i>Food hedonics</i>		
Explicit liking		
Explicit wanting		
Implicit wanting		
<i>Psychometric traits</i>		
Restraint		
Disinhibition		
Hunger		
BAS		
BIS		
STR		
BES		

## 9.7 Discussion II

The final aim of the current study was to assess whether there was an association between theoretically relevant common gene variants with eating behaviour, body composition and psychometric traits (intermediary phenotypes) that characterise trait binge eating.

### 9.7.1 Reward related genes

Previous research has shown that opioid peptides have a specific role in mediating the oro-sensory reward properties of food with opioid antagonists reducing the hedonic response to, and intake of palatable foods (Drewnowski et al., 1995; Martin R Yeomans & Gray, 2002; Ziauddeen et al., 2012). From this it was hypothesised that variation in two OPRM1 SNPs, rs1799971 and rs495491 would be associated with an increased intake of, and liking for high-fat foods. The hypotheses were not

supported and variation at rs1799971 and rs495491 was not associated with any of the variables measured in the current study.

When the influence of the DRD2 related rs1800497 SNP was examined, it was shown that individuals in the A1+ group had a significantly higher BMI compared to individuals in the A1- group. This finding was consistent with previous reports (Blum et al., 1996; Chen et al., 2012). Previous research has suggested that the presence of the A1 allele is associated with reduced brain dopamine D2 receptor availability that may contribute to a hypo-functioning reward system (Noble et al., 1991; Ritchie & Noble, 2003; Thompson et al., 1997). In line with this, the presence of the A1 allele has been implicated with increased risk of several substance abuse disorders such as alcoholism (Munafo, Matheson, & Flint, 2007) and heroin addiction (Xu et al., 2004). With regards to energy intake and obesity, Epstein, Temple, et al. (2007a), in line with previous research, found that energy intake and food reinforcement were greater in obese individuals compared to non-obese individuals, however within the obese individuals, food reinforcement and energy intake were greatest for those with at least one copy of the A1 allele. Further to this, in an fMRI study, Stice, Spoor, Bohon, Veldhuizen, and Small (2008) found that compared to lean individuals, obese individuals had a blunted striatal response to the ingestion of palatable food. Importantly, this effect was moderated by Taq1 status, with a stronger blunting of striatal activation observed in those with at least one copy of the A1 allele (Stice, Spoor, et al., 2008). These findings complement the view that a hypo-functioning reward system may arise as a consequence of overeating. They also suggest that down-regulation of the reward circuitry may be more severe for those who have a possible genetic predisposition to a hypo-functioning reward system (via the rs1800497 A1 allele). In the current study, variation at rs1800497 was not associated with food hedonics. However, when we examined the association between Taq1 genotype and implicit wanting in the Leeds sample we found that individuals in the A1- group had significantly higher implicit wanting for high-fat

sweet foods compared to individuals in the A1+ group (see Appendix 10 for figure and outcome of the analysis). While any interpretation of this finding needs to be considered with caution, it does appear to be consistent with evidence showing that the A2/A2 genotype is associated with an enhanced preference for highly palatable foods (i.e. those high in fat and sugar) (Davis et al., 2009; Davis et al., 2012). When the association between rs1800497 and psychometric traits was examined, it was shown that individuals in the A1+ group had lower scores on the Fun Seeking and Reward Responsiveness subscales of the BAS compared to those in the A1- group. However, it should be noted that the differences in scores between the groups were very small.

Variation at rs6277 has been related to dopamine D2 binding potential in the striatum, with the highest binding observed in the homozygous T genotype and the lowest in the homozygous C genotype (Hirvonen et al., 2004; Hirvonen et al., 2005). The findings from the current study demonstrated that compared to individuals with the CC genotype, those with the CT genotype consumed more energy, and exhibited a greater preference for sweet foods in the ad libitum eating task. In addition, there was a trend for the same pattern of consumption in individuals with the TT genotype. This finding is in accordance with Eny, Corey, and El-Sohemy (2009) who examined the effect of the rs6277 on habitual sugar consumption. They found that individuals with the CT genotype had a greater habitual sugar intake per day compared to individuals with the CC genotype. Taken together, these findings tentatively suggest that variation at rs6277 may influence food preferences, although further corroboration is warranted.

Further to this, individuals with the CC genotype had a higher BMI and waist circumference compared to those with the CT or TT genotype. In addition, in participants who underwent air plethysmography, the CC genotype was associated with a significantly greater percentage of body fat compared to the CT genotype. These findings were unexpected and in contrast to the energy intake findings



discussed above. In addition, to our knowledge this is the first report of an association between rs6277 and measures of adiposity. For these reasons and due to the small sample size and heterogeneity of the current sample this finding should be interpreted with caution.

### **9.7.2 Taste related genes**

The sensory aspects of food play a pivotal role in food selection and have been demonstrated to contribute to dietary preferences (Drewnowski et al., 2001; Glanz et al., 1998). The current study examined whether common variants in genes associated with the perception of sweet, bitter and fat taste were related to body composition, food choice and food preferences and food hedonics. It was hypothesised that variation in the TAS1R2 and SLC2A2 gene would be related to the increased intake of, and preference for sweet foods.

#### **9.7.2.1 Genes related to sweet taste**

Variation in rs35874116 of the TAS1R2 gene was associated with anthropometrics and body composition. Specifically, individuals with the TT genotype had a higher BMI, waist circumference, fat mass and percentage body fat compared to individuals with at least one copy of the C-allele. To our knowledge this has not been reported before. Contrary to the hypothesis, individuals with the TT genotype did not exhibit an enhanced preference for and intake of sweet foods. This finding was not consistent with previous research that has demonstrated that the TT genotype is associated with reduced sucrose sensitivity compared to the CC genotype (Fushan et al., 2009) and a higher habitual sugar intake (Eny et al., 2010).

Variation in the rs5400 variant of the SLC2A2 gene was associated with energy intake. Specifically, individuals in the T- group consumed a greater amount of energy from savoury foods compared to individuals in the T+ group. Further to this, there was a trend, which indicated that the T+ group had greater liking for sweet foods compared to the T- group. This finding was consistent with our hypothesis and tentatively supports previous research that has demonstrated individuals with the CC

genotype (T- group) reported habitually consuming less sugar compared to individuals with the CT or TT genotype (Eny et al., 2008).

#### **9.7.2.2 Genes relating to fat taste**

While preference for high fat food is thought to be universal, the strength of preference varies markedly between individuals and it has been suggested that increased sensitivity to fat may influence habitual fat consumption with potential consequences for body weight (Keller, 2012). The current study examined two variants in the CD36 gene, which previous research has shown may be associated with body composition, oral detection of fatty acids and a preference for foods containing high levels of added fat (Keller et al., 2012; Pepino et al., 2012). In the overall sample, individuals with the CC genotype of the rs2151916 polymorphism had significantly smaller waist circumference compared to individuals with the CT and the TT genotype. Further to this, in those individuals who underwent air plethysmography, individuals with the CC genotype of rs2151916 had significantly lower fat mass, BMI and waist circumference compared to individuals with the CT and the TT genotype. In addition, individuals with the GG genotype for rs1761667 had lower fat mass compared to individuals with the GA and the AA genotype. Importantly, the TT and TC genotypes of rs2151916, and the AA and GA genotypes of rs1761667 had body fat levels within the normal range expected for males and females, therefore, the effect seems to be that individuals with the CC genotype of rs2151916 and the GG genotype of rs1761667 possessed particularly low levels of fat mass and this was observed in both males and females across a range of BMIs. In addition, these effects appeared specific to markers of adiposity, as there were no differences in fat free mass between genotypes. Previous research examining the association between CD36 and body composition has been inconsistent to date with some studies supporting a link (Bokor et al., 2010; Heni et al., 2010) and others not (Choquet et al., 2010; Goyenechea et al., 2008).

Contrary to previous reports, the present study did not find an effect of rs2151916 or rs1761667 on food choice, food preferences or food hedonics. It is possible that the acute, cross-sectional nature of the behavioural measures employed in the present study were not sufficient to capture the small but persistent effects to be anticipated from common mutations on a single gene. Alternatively it is quite possible that the influence of CD36 on body composition is not mediated through behaviour but rather through an alternative mechanism such as fat metabolism, or monitoring of fat ingestion rather than having a direct effect on food choice and preference itself.

### **9.7.3 Obesity related genes**

Previous research has shown that variation in the FTO gene is associated with increased body weight, enhanced risk for obesity and increased intake of palatable foods (Cecil et al., 2008; Frayling et al., 2007; Hakanen et al., 2009; Wardle et al., 2008). In line with previous research the current study demonstrated that the AA and TA genotypes of rs9939609 were associated with a trend towards increased overall energy intake, and a significant increase in the intake of sweet snack foods compared to the TT genotype. Further to this, individuals with the CT genotype of rs1121980 consumed significantly more energy overall compared to individuals with the CC genotype. In addition, the presence of the T allele was associated with greater intake of sweet snack foods. However unlike previous research, variation in either rs9939609 or rs1121980 was not associated with BMI or body composition. It is important to note that while the presence of the FTO risk alleles may enhance susceptibility to obesity, they do not present a biological inevitability. Indeed research has demonstrated that physical activity is able to attenuate the effect of the FTO rs9939609 and rs1121980 genotype on BMI (Li et al., 2010; Vimalleswaran et al., 2009).

Based on previous findings that common variants in the MC4R gene have been associated with increases in body weight and a greater tendency to engage in binge eating behaviour (Branson et al., 2003; Loos et al., 2008; Qi et al., 2008) it was

hypothesised that variation at rs17782313 would be associated with greater energy intake and differences in body composition. This hypothesis was not supported and variation at rs17782313 was not associated with any of the variables measured in the current study.

## **9.8 General Discussion**

The first aim of the current study was to examine whether a common underlying genotype could be identified for the trait binge eating behavioural phenotype that has been characterised in the previous chapters of this thesis. In order to do this, the study adopted a candidate gene approach in which genes of interest were identified from a literature search to find SNPs that have previously been studied in relation to binge eating, preferences for foods high in sugar or fat, and obesity. The genotype frequency of the examined SNPs did not differ between high and low scorers on the Binge Eating Scale, therefore it was concluded that with regards to the SNPs examined, and there was no common underlying genotype for the trait binge eating phenotype.

Interestingly, the findings from the ad libitum eating task suggested that the tendency to binge eat in males may be quantitatively and qualitatively different to the tendency to binge eat in females as the preference for high fat sweet foods reported in the previous chapters was only found in female high scorers. This finding was consistent with previous research that has demonstrated that compared to males, females with the tendency to binge eat are more likely to endorse experiencing a loss of control over eating (Striegel-Moore et al., 2009) and are more likely to report eating snack based comfort foods (Wansink et al., 2003).

The second aim of the current study was to examine the association of the identified theoretically relevant SNPs with eating behaviour, body composition and psychometric traits that appear to characterise trait binge eating (intermediary phenotypes). Seventeen SNPs across thirteen genes were selected from a literature

search and were categorised according to their relevant function to create three groups; reward related genes, taste related genes and obesity related genes.

With regards to eating behaviour, the current study demonstrated, in line with previous research, that the FTO rs9939609 and rs1121980 polymorphisms were associated with the overall energy intake and energy intake from sweet foods (Cecil et al., 2008; Frayling et al., 2007). In addition, the findings of the current study supported a previously reported association between the DRD2 rs6277 CT genotype and increased habitual sugar intake (Eny et al., 2009), as it was found that compared to the CC genotype, individuals with the CT and the TT genotype consumed more energy overall and exhibited a greater preference for sweet foods in the ad libitum eating task. Finally, individuals with the SLC2A2 T- genotype appeared to have a greater preference for savoury foods compared to individuals with the T+ genotype. Further to this, the T+ group had greater explicit liking ratings for sweet foods compared to the T- group. While not entirely consistent with, these findings complement previous research that has shown that the presence of the T allele is associated with greater habitual sugar consumption (Eny et al., 2008).

Of the seventeen SNPs examined, five were associated with anthropometrics and body composition. In line with previous research, individuals with at least one copy of the ANKK1 A1 allele had a greater BMI compared to those who were A2 homozygous (Blum et al., 1996; Chen et al., 2012). In addition, it was demonstrated that individuals with the CC genotype, and individuals with the GG genotype of the CD36 rs2151916 and rs1761667 polymorphisms, respectively had lower BMIs and levels of body fat when compared to the other genotypes. To date, previous research examining the association between CD36 and body composition has been inconsistent (Bokor et al., 2010; Choquet et al., 2010; Goyenechea et al., 2008; Heni et al., 2010). While the effect in the present study appeared to be strong – there was an 8kg difference in fat mass between the CC genotype and the CT and TT genotype of rs2151916 – no mechanism was identified, as the genotypes did not differ on the

other outcome measures. Therefore, further investigation is required for confirmation and for identification of a potential mechanism for this effect.

Unexpectedly, it was found that individuals with CC genotype of the DRD2 rs6277 polymorphism had a higher BMI and a larger waist circumference compared to individuals with the CT or the TT genotype. This finding stood in contrast with the energy intake data in which individuals with the CC genotype consumed fewer calories compared to those with the CT or the TT genotype. These inconsistencies may be due to the cross-sectional design of the study in which it may not be expected for the outcomes from the ad libitum eating task and body composition to be complementary. It could be argued that the effect on body composition is more reliable as it is a more enduring characteristic of the individual that is not vulnerable to design limitations. In addition, it was found that individuals with the TT genotype of TAS1R2 rs35874116 had a higher BMI, waist circumference, fat mass and percentage body fat compared to individuals with at least one copy of the C-allele. It is important to note that the associations with body composition and DRD2 rs6277, and TAS1R2 rs35874116, do not appear to have been reported previously. Therefore, in light of the heterogeneity of the relatively small sample used in the current study it is important to interpret these findings with caution, as they need to be corroborated in a larger, better defined sample.

The present study had some limitations that should be considered. The sample used in the current study was arguably too heterogeneous for the results not grounded in previous research to be considered reliable and would have benefitted from being more defined in terms of BMI range and ethnicity. Furthermore, candidate gene association studies should ideally be conducted in more than one (well-defined) study population. Further to this, it may have been preferable to measure food hedonics and energy intake in a fed rather than a fasted state, which may have been more sensitive to detecting differences in susceptibility to reward-driven eating (Lowe, 2013). With regards to the first aim a case-control design, with participants

recruited on the basis of their binge eating score and matched by age and BMI to a control, would have been stronger. As discussed previously, the tendency to binge eat may be characterised by a distinct subset of behaviours in males and females which may suggest that there is a need for them to be examined separately. Finally, the sample in the current study was recruited from two distinct populations in the UK and three independent researchers conducted the study. This may have resulted in an accumulation of experimental noise and reduced level of sensitivity in the study.

In summary, the current study suggests that a common genotype may not underlie the trait binge eating phenotype as characterised in previous chapters. However, genetic markers were found for some intermediary phenotypes with regards to eating behaviour and body composition.

## **9.9 Summary**

- The genotype frequency of the examined gene variants did not differ between high and low scorers on the Binge Eating Scale, suggesting that a simple common genotype may not underlie the trait binge eating phenotype.
- The tendency to binge eat in males appeared to be quantitatively and qualitatively different to the tendency to binge eat in females, with the increased consumption of, and preference for sweet foods only evident in females.
- Common polymorphisms in the FTO, DRD2 and SLC2A2 genes were associated with eating behaviour. For the FTO gene, the presence of the A allele of the rs9939609 polymorphism and the presence of the T allele of the rs1121980 polymorphism was associated with an increased preference for sweet foods. For the DRD2 gene, individuals with the CT and TT genotypes of rs6277 consumed more energy overall and exhibited a greater preference for sweet food compared to those with the CC genotype. Finally, for the SLC2A2 gene, individuals in the T+ group consumed less energy from

savoury foods and had greater explicit liking ratings for sweet foods compared to those in the T- group.

- Common polymorphisms in the ANKK1, DRD2, TAS1R2 and CD36 genes were associated with anthropometrics and body composition. In line with previous research, the A1 allele of the ANKK1 rs1800497 polymorphism was associated with higher BMI. Furthermore, the rs1761667 and rs2151916 variants in the CD36 gene were associated with lower BMI and levels of fat mass. In addition, it was demonstrated that individuals with the CC genotype of the DRD2 rs6277 polymorphism and individuals with the TT genotype of the TAS1R2 rs35874116 had a higher BMI compared to the other genotypes. However, these findings have not been reported previously and require confirmation in a larger sample.



## General Discussion

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### 10.1 Overview of studies

The research presented in this thesis is the culmination of experimental studies designed within a biopsychological framework to examine the role of liking and wanting for food as risk factors for overeating in trait binge eating, and to determine other potential markers of susceptibility (including physiological and genetic) in this phenotype. To see a general overview of the findings in this thesis please see Table 10.1 p.219.

#### 10.1.1 Comparing measures of food wanting

The first study was designed with two parallel objectives, which were presented separately in Chapters 5 and 6. The first objective addressed two primary aims, the first of which was concerned with comparing two behavioural measures of the non-verbal motivational component of reward – food wanting – to determine whether they measured a shared underlying process. To do this, participants completed two commonly used, reaction-time based measures of food wanting – attentional bias, assessed using the modified Visual Probe Task (VPT) and implicit wanting, assessed using the Leeds Food Preference Questionnaire (LFPQ), under counterbalanced fasted and fed conditions. The second aim was concerned with examining whether each measure of food wanting was sensitive to differences in motivational state and predictive of ad libitum snack food intake in order to determine whether one measure was more valid and versatile than the other and could therefore be used in subsequent studies in this thesis. The results indicated that the VPT and the LFPQ appeared to assess a dissimilar underlying process as the implicit wanting measure of the LFPQ was not associated with either the 100ms or the 500ms attentional bias measures of the VPT. It was suggested that the lack of a meaningful associations might have resulted due to key methodological differences between the two

measures. Specifically, the LFPQ utilises a forced choice paradigm in which participants are presented with a series of food choices from which they must select which food item they most want to eat now. This ease with which this choice is made (which is reflected in the relative category response times) is dependent on the hedonic value of the food images, therefore the choice made may be more proximal to the behaviour of food intake (a suggestion that was supported by the associations between implicit wanting and energy intake). In comparison, the VPT assesses the ‘attention grabbing’ properties of the food images, which in the absence of choice, may be more distal to the behaviour of food intake (a suggestion supported by the absence of association between attentional bias and energy intake). Therefore, attentional bias for food might be a more elusive component of food-related motivation, rather than being a strong independent determinant of actual eating behaviour.

With regards to the second aim, the findings from the LFPQ were in accordance with previous research, as participants’ explicit ratings were lower in the fed compared to the fasted condition, whereas, implicit wanting for sweet foods was greater in the fed compared to the fasted condition. The findings from the VPT revealed that participants had a greater attentional bias for food images relative to non-food images in both the fasted and fed condition for the orientation of attention trials, and in the fasted condition for the maintenance of attention trials. Only the latter finding was consistent with the hypothesis and it was suggested that the calculation of an overall attentional bias for food-stimuli might not be as sensitive when the food-images are manipulated to try and capture specific food category biases. While the results from the maintenance of attention trials were consistent with the literature on sensory-specific satiation neither the orientation nor the maintenance measures of attentional bias appeared sensitive to detecting individual differences in food wanting (assessed in Chapter 6). Therefore, the implicit wanting measure of the

LFPQ was used in subsequent studies as it appeared to be a more sensitive, feasible and flexible measure of food wanting.

### **10.1.2 The effect of trait binge eating in normal weight females**

The second objective of the first study, presented in Chapter 6, had two primary aims. The first was to determine whether trait binge eating was a psychobiological marker for susceptibility to overeating in a sample of normal weight females. The second was to examine the influence of trait binge eating on liking and wanting for food, and food intake in an ad libitum eating task. Participants were categorised as either lean 'binge-type' (L-B) or lean 'non-binge type' (L-NB) based on their scores on the Binge Eating Scale. Liking and wanting for food was assessed under counterbalanced fasted and fed conditions, whereas food intake was assessed while participants were in a fed state. There was no effect of BES on subjective ratings of appetite sensations. Higher BES scores were associated with psychometric traits, body composition, food choice and food hedonics. Specifically, compared with L-NB, L-B scored higher in trait disinhibition and trait hunger, a combination of traits, which may suggest they are more vulnerable to opportunistic eating (Blundell et al., 2005). Further to this, L-B had a significantly higher percentage body fat compared to L-NB. When the influence of trait binge eating on food hedonics was examined, it was shown that L-B appeared to have stronger, persistent liking for food that was similar in the fasted and the fed condition. Whereas, L-NB's liking for food was lower in the fed compared with the fasted condition. Further to this, L-B had greater implicit wanting, specific for high-fat sweet foods, under fasted and fed conditions compared to L-NB. This enhanced implicit wanting for high-fat sweet foods was in accordance with the findings from the ad libitum eating task, as while there were no differences between L-B and L-NB in overall energy consumed, L-B consumed 40% more sweet foods. Taken together, these findings demonstrate that trait binge eating is a psychobiological marker for susceptibility to reward-driven eating that is characterised by enhanced liking for food overall, and greater implicit wanting for (and intake of) high-fat sweet foods, specifically. These findings also highlighted

that trait binge eating appears to be functional at relatively low to moderate levels in a normal weight, non-clinical population.

### **10.1.3 The effect of trait binge eating in overweight or obese females**

The primary aims of the second study were to 1) determine whether trait binge eating was a psychobiological marker for susceptibility to reward-driven overeating in overweight or obese females and 2) examine the role of liking and wanting as risk factors for overeating. A secondary objective was to further examine the influence of trait binge eating on liking, wanting, food intake and food choice in normal weight individuals. Using a matched pairs design, normal weight and overweight or obese participants were categorised, as either 'binge-type' or 'non-binge type' based on their scores on the BES to create four groups; obese binge-type (O-B), obese non-binge type (O-NB), lean binge-type (L-B) and lean non-binge type (L-NB). In a refinement of the food intake methodology used in the first study, the ad libitum eating task was presented in both the fasted and the fed condition, and a sweet component was added to the fixed energy test meal. Further to this, to accommodate individual differences in energy requirements, the fixed energy test meal was calibrated to provide participants with 25% of their individual daily energy requirements.

Consistent with previous research (Epstein, Temple, et al., 2007b; Nijs et al., 2010; Saelens & Epstein, 1996), age-matched overweight or obese females consumed more energy ad libitum under fasted and fed conditions compared to their lean counterparts. However, when individual differences in binge eating behaviour were taken into consideration, it was shown that only O-B consumed more energy compared to O-NB. In line with the findings from Chapter 6, both O-B and L-B exhibited a greater preference for sweet foods, and higher levels of opportunistic eating traits. Further to this, compared to O-NB, O-B had an enhanced liking for food overall and greater implicit wanting for high-fat sweet foods that appeared to be independent of their motivational state. Concurrently, binge-eating score was positively associated with self-reports of craving intensity and experience of craving

for sweet foods over the previous seven days. Furthermore, binge-eating tendency was associated with measures of central adiposity as O-B had a larger waist circumference compared to O-NB – a finding that does not seem to have been previously reported in the literature. In the normal weight groups, the findings were mostly consistent with the previous study, as L-B had enhanced implicit wanting for high-fat sweet foods in the fed condition compared to L-NB. However, unlike the findings of Chapter 6, L-B had enhanced implicit wanting for low-fat sweet foods (rather than high-fat sweet foods) in the fasted condition. This raised the question as to whether trait binge eating, in normal weight individuals, was characterised by a preference for high-fat sweet foods specifically, or for sweet taste in general. With regards to the second aim, liking and wanting for were associated with food intake in the fasted and fed condition. Interestingly however, the association between explicit ratings and food intake were stronger in the fed condition; this finding is consistent with the notion that in the absence of hunger, an enhanced hedonic response to food may be a risk factor for increased consumption. The association between food intake and the implicit wanting measure did not follow the same pattern; however under circumstances in which participants are explicitly informed to consume as much or as little as they would like it might be expected that explicit processes have a more dominant role in eating behaviour. In summary, the findings of Chapter 7 provided evidence to support that trait binge eating forms a distinct behavioural phenotype in overweight or obese individuals that was characterised by differences in central adiposity, liking and wanting for food, energy intake, food choice and the experience of food cravings.

#### **10.1.4 Is trait binge eating an ecologically valid phenotype?**

The third study, presented in Chapter 8, had two main objectives. The first was concerned with examining whether the behaviours and processes observed under laboratory conditions in O-B and L-B extended to eating behaviour in their natural setting. In a matched pairs design, overweight or obese females, and a smaller subset of normal weight females, were recruited on the basis of their score on the BES to

form the four groups used in Chapter 7. Energy intake was assessed over two 24-hour periods using combined laboratory-based test meal methodology and free-living dietary recall procedures. Consistent with the findings from the second study, under laboratory conditions, O-B consumed more energy overall from snack foods compared to O-NB and exhibited a greater preference for high-fat sweet foods, and reported experiencing greater craving intensity and cravings for these kinds of foods over the previous seven days. Furthermore, compared to O-NB, O-B had greater liking for high-fat foods, and greater implicit wanting for high-fat sweet foods under both fasted and fed conditions. When 24-hour laboratory energy intake was analysed it was shown that compared with O-NB, O-B consumed significantly more than their estimated energy requirements. Interestingly, however there was evidence for overconsumption in all groups under laboratory conditions. A similar pattern of overconsumption was not observed from the free-living based measure of energy intake, for which energy intakes were more in line with estimated daily energy requirements. However, some findings were consistent as under free-living conditions, O-B reported consuming a greater number of sweet snack foods compared to O-NB and there was a trend for O-B to report consuming a greater amount of energy overall from snack foods. It was suggested that the differences between laboratory-based and free-living based measures of energy intake might have arisen due to some methodological limitations. However, while the outcomes of the laboratory-based and free-living based measure of energy intake were not identical with regards to total energy consumed, they did provide a coherent view of trait binge eating – with greater intake of and preference for high-fat sweet foods evident across both measures. Furthermore, it is evident from the content of the dietary recalls from O-B (see Appendix 7) that the increased preference for sweet foods is salient in their eating behaviour throughout the day – and is not limited to snacking behaviour. Therefore, these data provide evidence that trait binge eating identifies an ecologically valid phenotype of obesity.

#### **10.1.5 Does ‘food addiction’ form a distinct behavioural phenotype?**

The second aim of the third study was concerned with whether the construct of ‘food addiction’, defined by Yale Food Addiction Scale (YFAS; Gearhardt et al., 2009), was able to identify a subtype of disordered eating that was distinct from the binge eating phenotype. In order to examine this aim, O-B were divided into ‘food addicts’ (n=4) and ‘non-food addicts’ (n=8) based on the diagnostic threshold for food addiction defined by Gearhardt et al., (2009). It was demonstrated ‘food addicts’ had a larger waist circumference and greater levels of fat mass compared to ‘non-food addicts’. Further to this, ‘food addicts’ consumed more energy overall under laboratory conditions, and more energy from snack foods under both laboratory and free-living conditions. The differences between ‘food addicts’ and ‘non-food addicts’ remained significant even when differences in estimated energy requirements were controlled for. Further to this, the ‘food addicts’ appeared to be partly distinct from O-B as a whole as they exhibited a preference for both sweet and savoury high-fat snack foods under laboratory conditions. ‘Food addicts’ also appeared to have a greater level of eating pathology as they had higher scores on both trait disinhibition and binge eating compared to ‘non-food addicts’. While it is important to highlight the very low number of individuals meeting the YFAS criteria for food addiction, these preliminary results suggested that ‘food addiction’, as defined by the YFAS, appears to fit along a continuum of the BES and may therefore correspond to a more severe expression of binge eating tendency rather than being behaviourally distinct from it.

#### **10.1.6 Laboratory based versus free-living based measures of eating behaviour**

Measuring eating behaviour under laboratory-based and free-living based conditions allowed for the associations between the two measures of energy intake to be examined. The outcomes were promising, as there was a strong correlation between overall energy intake from both measures and between laboratory-based and free-living based snack intake – a finding which supported the validity of the ad libitum eating task as a measure of energy intake and food choice. Further to this, there was

a negative relationship between implicit wanting for low-fat savoury foods and overall energy intake, and snack food intake under free-living conditions. This finding was repeated throughout the thesis and was also evident in the studies presented in Chapters 5 and 7. It can be suggested that greater implicit wanting for low-fat savoury foods may be a marker of resistance to excessive consumption not only of high-fat snacks (such as the ones used in the ad libitum eating task) but also over-consuming in the free-living environment. These findings are intriguing because they suggest that implicit wanting for food per se is not a risk factor for overconsumption, the direction of this motivation is important. Furthermore, this relationship was only observed in the more covert measure of implicit wanting, and was not apparent in the explicit ratings.

#### **10.1.7 Examination of potential genetic markers for susceptibility to overeating**

The final study, presented in Chapter 9, had two primary objectives. The first objective was to determine whether a common underlying genotype could be identified for the behavioural trait binge eating phenotype characterised in the previous studies. The second objective was to examine the association between relevant genetic markers and the characteristics that findings from this thesis have identified to be associated with trait binge eating (so-called ‘intermediary phenotypes’). Seventeen single nucleotide polymorphisms (SNPs) within thirteen genes were identified and categorised according to their relevant genetic function; reward related genes, taste related genes and obesity related genes. With regards to the first objective, there appeared to be no common underlying genotype in the examined SNPs for the trait binge phenotype. However, the study would have benefitted from using a case-control design, in which participants were recruited on the basis of their binge eating score and matched by age and BMI to a control (e.g. Davis, Levitan, Carter, et al., 2008).

When the intermediary phenotypes were examined, four of the seventeen SNPs identified were related to eating behaviour, five were associated with



anthropometrics and body composition, and two were associated with liking and wanting. In accordance with previous research, two common polymorphisms (rs9939609 and rs11211980) in the FTO gene, and one in the DRD2 gene (rs6277) were associated with an increased intake of sweet foods in the ad libitum eating task (Cecil et al., 2008; Eny et al., 2009). Further to this, variation in the rs5400 polymorphism of the SLC2A2 was associated with differences in food preferences. Individuals with the CC genotype consumed more energy from savoury foods compared to individuals with the CT and TT genotype. Conversely, individuals with the CT and TT genotype had higher explicit liking ratings for sweet foods compared to those with the CC genotype. Although this was not associated with increased intake of sweet foods it did complement previous research which has shown that individuals with the CT and TT genotype reported consuming a greater amount of sugar in their habitual diets (Eny et al., 2008). Interestingly, the A2/A2 genotype was associated with greater implicit wanting for high-fat sweet foods, a finding that is consistent with previous research (Davis et al., 2009; Davis et al., 2012). However, while intriguing, this association was only evident in the Leeds sample and therefore further corroboration in a larger better-defined sample is required. With regards to anthropometrics and body composition, evidence was found to support previous research that the presence of the A1 allele is associated with an increase in BMI (Blum et al., 1996; Chen et al., 2012). Further to this, variation in two common polymorphisms (rs1761667 and rs2151916) in the putative fat taste receptor gene, CD36, appeared to be associated with low levels of fat mass however to date, previous research examining variation in CD36 and body composition has been inconsistent (Bokor et al., 2010; Choquet et al., 2010; Goyenechea et al., 2008; Heni et al., 2010). Finally, variation in the TAS1R2 gene and the DRD2 gene were unexpectedly associated with measures of body composition. However, as these findings have not been reported previously, and due to the heterogeneity of the current study sample they require confirmation in a larger sample.

#### **10.1.8 Do the behaviours and processes underlying trait binge eating in females apply to males?**

In addition to examining potential genetic markers for trait binge eating, the final study allowed for the examination of trait binge eating in males. The findings revealed interesting gender differences in the binge eating phenotype as it appeared that the tendency to binge eat in males was quantitatively and qualitatively different to the tendency to binge eat in females, with the increased preference for, and consumption of sweet foods only evident in females. This finding does not appear to have been reported before, however it does appear to be consistent with the literature examining gender differences in comfort food preferences (Wansink et al., 2003).

#### **10.1.9 The trait binge eating phenotype**

The research presented in this thesis has provided evidence that trait binge eating appears to be a distinct, ecologically valid, behavioural phenotype of obesity that is characterised by enhanced liking and wanting for food, a greater preference and craving for sweet foods under both laboratory and free-living conditions, lower reports of positive mood and differences in measures of adiposity. Further to this, both O-B and L-B had greater scores on the trait disinhibition and trait hunger subscales of the TFEQ when compared to O-NB and L-NB, respectively. This finding suggests that the trait binge eating phenotype is complex and encompasses both the tendency to eat to excess (assessed by greater BES scores) and the tendency to eat opportunistically (assessed by a combination of high levels of trait disinhibition and hunger). In order to better understand the composite predictors of the behavioural tendencies observed in the phenotype (e.g. increased preference for high-fat sweet foods and greater implicit wanting for high-fat sweet foods) multiple regression analyses were conducted with trait binge eating, trait disinhibition and trait hunger entered as predictors (see Appendix 11 for the outcome of this analysis). The analysis revealed that trait binge eating was the strongest predictor of increased preference for sweet foods under both laboratory (Chapter 7 and Chapter 8) and free-living conditions (Chapter 8) and was the only variable to significantly explain any

unique variance in this model. Further to this, trait binge eating was the strongest predictor of implicit wanting for high-fat sweet foods (Chapter 8) and similarly, was the only variable to significantly explain any unique variance in this model. Therefore, trait disinhibition and trait hunger can be viewed as composite vectors within the trait binge eating phenotype which contribute to overeating.

The results of Chapter 6 demonstrated that a savoury test meal resulted in a decrease in ratings of explicit liking for savoury foods in both L-B and L-NB, a finding consistent with the literature on sensory specific satiation. Future research may examine the impact of trait binge on sensory specific satiation with a predominantly sweet tasting test meal as previous evidence suggests that this may further stimulate implicit wanting for sweet foods and perhaps increase intake of sweet foods (Finlayson, Bordes et al., 2012). Further to this, the binge eating phenotype did not show differences in appetite ratings throughout Chapters 5 to 9 or in the satiating efficiency of either a fixed energy test meal (Chapter 7) or an ad libitum test meal (Chapter 8). Therefore, the trait binge eating phenotype does not appear to be characterised by differences in measures of satiety.

It must be noted that the findings in normal weight individuals were to some degree inconsistent, as in the first study L-B appeared to exhibit greater implicit wanting for high-fat sweet foods, whereas in the second study they appeared to exhibit greater implicit wanting for low-fat sweet food. Furthermore, under free-living conditions there appeared to be a non-significant trend for L-B to consume more energy from both low-fat and high-fat sweet foods compared to L-NB. Therefore it is not apparent whether L-B are characterised by enhanced implicit wanting for high-fat sweet foods specifically, or for sweet taste in general. However, based on findings from previous research, it can be suggested that implicit wanting for sweet tasting foods may have implications for appetite control (de Graaf, Schreurs, & Blauw, 1993; Finlayson, Bordes, et al., 2012). However, in overweight or obese individuals,

trait binge eating seems to be a distinct phenotype with reliable psychological and anthropometric characteristics.

Table 10.1 Summary of the aims and main findings from the experimental chapters of the thesis

	<b>Chapter 5</b>	<b>Chapter 6</b>	<b>Chapter 7</b>	<b>Chapter 8</b>	<b>Chapter 9</b>
<i>Study aim(s)</i>	<ul style="list-style-type: none"> <li>• To compare two measures of wanting – VPT and LFPQ.</li> <li>• To determine whether measures on VPT and LFPQ were associated with energy intake.</li> <li>• To examine how the VPT and LFPQ performed under fasted or fed motivational states.</li> </ul>	<ul style="list-style-type: none"> <li>• To examine the influence of trait binge eating on liking and wanting, food choice and energy intake in normal weight females.</li> <li>• To examine differences in body composition underlying variation in trait binge eating.</li> </ul>	<ul style="list-style-type: none"> <li>• To examine differences in liking and wanting, food choice and energy intake in relation to trait binge eating and body mass index.</li> </ul>	<ul style="list-style-type: none"> <li>• To determine whether the previous findings in overweight or obese individuals with greater trait binge eating scores extended beyond the laboratory situation and relate to free-living eating behaviour.</li> </ul>	<ul style="list-style-type: none"> <li>• To assess whether there was a common profile of genetic markers for the binge eating phenotype.</li> <li>• To examine the association of theoretically relevant common gene variants with eating behaviour, body composition and psychometric traits implicated in trait binge eating.</li> </ul>
<i>liking and wanting for food</i>	<ul style="list-style-type: none"> <li>• Consistent with sensory specific satiation, ratings of liking for savoury foods decreased following a savoury</li> </ul>	<ul style="list-style-type: none"> <li>• L-B's overall liking ratings for food were similar in the fasted and the fed condition, whereas L-NB's overall liking ratings</li> </ul>	<ul style="list-style-type: none"> <li>• L-B had greater liking ratings for high-fat sweet foods compared to L-NB.</li> <li>• L-B had greater implicit wanting for</li> </ul>	<ul style="list-style-type: none"> <li>• O-B had greater liking ratings for food overall compared to O-NB.</li> <li>• O-B had greater implicit wanting for</li> </ul>	<ul style="list-style-type: none"> <li>• Greater trait binge eating scores were associated with greater implicit wanting for high-fat sweet foods.</li> </ul>

<p>test meal, whereas ratings of liking for sweet foods remained stable.</p> <ul style="list-style-type: none"> <li>• Implicit wanting for savoury foods decreased following a savoury test meal, in contrast implicit wanting for sweet foods increased.</li> </ul>	<p>decreased in the fed condition.</p> <ul style="list-style-type: none"> <li>• L-B had greater implicit wanting for high-fat sweet foods in the fasted condition compared to L-NB.</li> </ul>	<p>high-fat sweet foods in the fed condition compared to L-NB.</p> <p>O-B had greater liking ratings for food overall compared to O-NB.</p> <ul style="list-style-type: none"> <li>• O-B had greater implicit wanting for high-fat sweet foods, specifically compared to O-NB in the fasted and fed condition.</li> <li>• Implicit wanting for low-fat savoury was negatively associated with overall energy intake.</li> </ul>	<p>high-fat sweet foods, specifically compared to O-NB in a fasted and a fed state.</p> <ul style="list-style-type: none"> <li>• Implicit wanting for low-fat savoury was negatively associated with overall energy intake under laboratory conditions, and energy consumed from fat and carbohydrate under free-living conditions.</li> </ul>	
Chapter 5	Chapter 6	Chapter 7	Chapter 8	Chapter 9
<i>Food choice and energy intake</i>				
<ul style="list-style-type: none"> <li>• Greater implicit wanting for sweet food was associated with greater overall energy intake and greater intake of sweet foods.</li> </ul>	<ul style="list-style-type: none"> <li>• L-B exhibited a greater preference for sweet snack foods compared to L-NB.</li> </ul>	<ul style="list-style-type: none"> <li>• L-B exhibited a greater preference for sweet snack foods compared to L-NB.</li> <li>• O-B exhibited a greater preference for</li> </ul>	<ul style="list-style-type: none"> <li>• O-B exhibited a greater preference for, and consumed more energy from, sweet snack foods under both laboratory and free-living</li> </ul>	<ul style="list-style-type: none"> <li>• In females, greater binge eating scores were associated with increased consumption of, and preference for sweet snack foods.</li> </ul>

<ul style="list-style-type: none"> <li>• Greater explicit liking for sweet food was associated with greater overall energy intake and greater intake of sweet foods.</li> </ul>	<p>sweet snack foods compared to O-NB.</p> <ul style="list-style-type: none"> <li>• While overweight and obese individuals consumed more energy than lean individuals, when trait binge eating was taken into consideration, only O-B consumed more energy overall.</li> </ul>	<p>conditions.</p>	<ul style="list-style-type: none"> <li>• In males, there were no differences in energy intake or food choice between the binge type and the non-binge type groups.</li> </ul>
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<i>Anthropometrics and body composition</i>
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<ul style="list-style-type: none"> <li>• L-B had greater percentage body fat than L-NB.</li> </ul>	<ul style="list-style-type: none"> <li>• O-B had a greater waist circumference compared to O-NB.</li> <li>• No differences between L-B and L-NB on measures of anthropometrics and body composition.</li> </ul>	<ul style="list-style-type: none"> <li>• O-B had a greater amount of fat mass and a greater waist circumference compared to O-NB.</li> </ul>
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Chapter 5	Chapter 6	Chapter 7	Chapter 8	Chapter 9
<i>Psychometric traits</i>	<ul style="list-style-type: none"> <li>• L-B scored higher on trait disinhibition and trait hunger compared</li> </ul>	<ul style="list-style-type: none"> <li>• L-B scored higher on trait disinhibition compared to L-NB.</li> </ul>	<ul style="list-style-type: none"> <li>• O-B scored higher on trait disinhibition and trait hunger compared</li> </ul>	<ul style="list-style-type: none"> <li>• Higher scores on trait binge eating were associated with higher</li> </ul>

	to L-NB.	<ul style="list-style-type: none"> <li>• O-B scored higher on trait disinhibition and trait hunger compared to O-NB.</li> </ul>	to O-NB.	trait disinhibition and trait hunger scores.
<i>Food cravings (COEQ)</i>				
		<ul style="list-style-type: none"> <li>• O-B reported greater craving intensity and greater cravings for sweet foods over the previous seven days compared to O-NB.</li> <li>• O-B reported experiencing lower levels of positive mood over the previous seven days compared to O-NB.</li> </ul>	<ul style="list-style-type: none"> <li>• O-B reported greater craving intensity and greater cravings for sweet foods over the previous seven days compared to O-NB.</li> <li>• O-B reported experiencing lower levels of positive mood over the previous seven days compared to O-NB.</li> </ul>	



## **10.2 Methodological Issues**

Behavioural studies on human appetite must make (and adhere to) an important distinction between the neuro-chemical mechanisms of liking and wanting derived from animal research models, and liking and wanting as psychological constructs in human beings, as one cannot infer that the latter is an interpretative read-out of the former. Indeed, a recent fMRI study examining the effect of an opioid antagonist on neural and behavioural measures of liking and wanting for high calorie compared to low calorie food images found that the neural and behavioural measures of motivation (or wanting) were not associated (Cambridge et al., 2013). To date, there is no standard or widely agreed metric for the assessment of the psychological constructs of liking and wanting in human appetite research, and verifying their operation in human eating behaviour is controversial (Finlayson & Dalton, 2012a; Havermans, 2012a, 2012b). The conceptualisation of liking and wanting as psychological constructs in the current thesis makes a move away

### **10.2.1 Are liking and wanting distinguishable in human eating behaviour?**

It is logical that the experience of reward involves a combination of liking and wanting and that both processes contribute to eating behaviour, for this reason one would hypothesise that subjective and behavioural measures of liking and wanting will be, to a certain degree, interrelated. However, by measuring these components separately it is possible to learn under which circumstances they may differ by degree, or even become dissociated, which may help to elucidate their role in susceptibility to overeating and to weight gain (Finlayson, Dalton, & Blundell, 2012; Finlayson et al., 2007).

For example, Griffioen-Roose et al., (2012) used the LFPQ to assess the impact of a 14-day dietary intervention on ad libitum food intake and food reward. They demonstrated that when participants were in a state of protein balance, liking and implicit wanting for food were similar. However, when participants were in a state of protein imbalance (as a result of a low-protein diet), implicit wanting was greater for

high-protein foods and appeared to exact a stronger determining role on what was eaten whereas there were no effect of condition on food liking. These findings suggest that liking for food may be relatively stable compared to implicit wanting, which appears to be more variable and under certain conditions enhanced for specific kinds of foods. The studies presented in Chapters 6, 7 and 8 of the current thesis provide evidence to support this suggestion as the overweight or obese binge-types had greater liking for food overall but they had enhanced implicit wanting for sweet foods, specifically.

The findings of the study presented in Chapter 5 provided tentative evidence that sensory specific satiation may be associated with differences in liking and wanting as following the savoury test meal, liking ratings for sweet foods decreased whereas implicit wanting for sweet foods increased. This effect has also been demonstrated in previous research (Griffioen-Roose et al., 2010; Griffioen-Roose, Mars, Finlayson, Blundell, & de Graaf, 2011). When incremental validity was assessed, using hierarchical multiple regression, ratings of liking and implicit wanting for high-fat sweet foods were entered as predictors of overall energy intake and sweet food intake (see Appendix 11), implicit wanting accounted for 14.9% unique variance in overall energy intake and for 10.4% unique variance in sweet food intake.

Finally, the findings from Chapters 5, 7 and 8 indicated that implicit wanting for low-fat savoury foods was negatively associated with snack food intake assessed under laboratory conditions, and overall energy intake and snack food intake under free-living conditions. This association tentatively suggests that 1) greater implicit wanting for food per se may not be a risk factor for overconsumption and 2) there may be a subtle disconnect between liking and implicit wanting. Taken together, these findings support the use of the LFPQ as a measure of food hedonics and highlight the value of distinguishing between liking and implicit wanting for food and for studying their role in eating behaviour.

## **10.3 Implications**

### **10.3.1 Binge eating and Binge eating disorder**

The recent publication of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) introduced several changes with regards to the diagnosis of eating disorders. Of relevance to the current thesis was the inclusion of BED from its DSM-IV category of a provisional eating disorder requiring further study, to a formally recognised eating disorder. The recognition of BED as a distinct eating disorder was accompanied by several changes to the criteria for its diagnosis, which included a reduction in the frequency of the required number of binge eating episodes from twice to once weekly and a reduction in the duration of binge eating behavior from six to three months. Therefore, under the new criteria a diagnosis of BED requires: 1) recurrent episodes of eating a larger amount of food over a short period of time than most individuals would consume under similar circumstances coupled with feelings of lack of control over eating; 2) binge eating episodes that are associated with at least three of – eating more rapidly than normal, eating until uncomfortably full, eating a large amount in the absence of hunger, eating alone due to feelings of embarrassment, and feeling disgusted, guilty or ashamed following the binge; 3) marked distress related to eating behavior; 4) binge episodes occur on average once per week over the course of three months; and 5) the binge eating is not followed by inappropriate compensatory behaviours (such as purging or use of laxatives). However, criticisms have been made regarding the change as it has been noted that the diagnosis of BED may not be distinguishable from non-pathologic forms of over-eating due to the lack of criteria for what defines an unusually ‘large amount of food’ and whether this relates to eating within and outside of meals (Stetka & Correll, 2013).

In the scientific literature, there has been a strong focus on binge eating as a clinical occurrence and the term ‘binge eating’ is often synonymous with BED. However, the estimated prevalence of BED in the general population is generally quite low and is estimated to be between 0.7-3.0% (Brownley et al., 2007; Hudson, Coit, Lalonde,

& Pope, 2012; Kessler et al., 2013) while recurrent episodes of binge eating, as a non-clinical occurrence, are estimated to occur in 10-20% of obese and lean individuals (Berg et al., 2009; Bruce & Wilfley, 1996; Spitzer et al., 1993; Striegel-Moore et al., 2009). The research in this thesis has provided evidence to support that binge eating, as a non-clinical occurrence, identifies a distinct behavioural phenotype of obesity that is characterised by greater liking for food overall and enhanced implicit wanting for high-fat sweet foods specifically, greater experiences of food cravings, lower reports of positive mood, higher levels of adiposity, and greater energy intake, especially of high-fat sweet foods under laboratory and free-living conditions. Further to this, binge eating has previously been associated with increases in fat mass over a period of one year in a sample of first year undergraduate students (Finlayson, Cecil et al., 2012). Indeed, the research in this thesis corroborates the association between fat mass and binge eating reported by Finlayson, Cecil et al. (2012) as the studies in Chapters 6, 7 and 8 showed that L-B and O-B had greater levels of adiposity (evidenced by differences in body composition or waist circumference) compared to L-NB and O-NB, respectively. Measures of adiposity have been reported to be better predictors of myocardial infarction than BMI (Yusuf et al., 2005) and may indicate that binge eating behaviour increases the risk for health problems which increased adiposity has been shown to predict (Zhu et al., 2002). Therefore, these findings highlight the importance of examining markers of adiposity in addition to BMI when defining obesity (and relevant subtypes). Furthermore, Stice et al., (2013) reported that subclinical levels of binge eating were associated with an enhanced risk of escalation to BED over a period of eight years in a sample of female adolescents (Stice et al., 2013). It can be tentatively suggested that the findings from Chapter 8, in which a small subset of O-B met the criteria defined by the YFAS for food addiction and exhibited higher levels of eating pathology, may represent a progression of binge eating severity. Therefore, binge eating appears to be a reliable psychobiological marker for susceptibility to overeating and weight gain and the characterisation of

the processes and behaviours that underlie trait binge eating may allow for the development of more tailored strategies to prevent potential escalation into more severe forms of disordered eating.

### **10.3.2 Liking and wanting as risk factors for overeating**

The findings from the current thesis suggest that the tendency to binge eat is underpinned by enhanced wanting in conjunction with greater liking for food that appear to be largely independent of motivational state. A reliable finding throughout the thesis was that O-B had increased implicit wanting for high-fat sweet foods when hungry compared to O-NB. Further to this, implicit wanting for high-fat sweet foods was associated with increased intake of these types of food under laboratory and free-living conditions. This enhanced implicit wanting for sweet foods in O-B may be marker for reduced appetite control and increased consumption and may be compounded by the high energy density of high-fat sweet foods and the apparent effect that sweet taste has on delaying satiation and subsequent food choices in individuals identified as being susceptible to reward-driven overeating. For example, Finlayson, Bordes, et al. (2012) examined the effect of a savoury or sweet tasting preload on energy intake in a sample of females who differed in their level of susceptibility to overeating (determined by their TFEQ disinhibition scores). They found that compared to the savoury tasting preload, energy intake in an ad libitum test meal increased following the sweet tasting preload in individuals scoring high in trait disinhibition. These findings suggested that in individuals susceptible to overeating, sweet taste might have a weaker modulating effect on satiation and subsequent food choice (de Graaf et al., 1993; Griffioen-Roose et al., 2010). Further to this, there was evidence to suggest that O-B exhibited greater implicit wanting for high-fat sweet foods compared to O-NB in the fed condition, and this finding was complemented by reports that they also experience greater food cravings for these types of foods. Therefore, it can be suggested that O-B are characterised by a persistent drive to eat these kinds of food, a suggestion that is partly supported by the food preferences observed in their free-living eating behaviour.

Previous research suggests that the homeostatic and hedonic systems of appetite control are affected by motivational state, and may operate in conjunction to promote or inhibit food intake (Berthoud & Morrison, 2008). Therefore, in the absence of internal need, food becomes less pleasurable as a consequence of it no longer being required to alleviate hunger (Cabanac, 1989). Consistent with this, the current thesis demonstrated that ratings of liking for all food categories were lower in the fed compared with the fasted condition. Interestingly, however, when the associations between ratings of liking and energy intake were examined, there was a stronger positive relationship between overall energy intake and liking for food in the fed compared to the fasted condition which suggests that greater liking for food in a fed state may be a risk factor for increased consumption. Altogether, these findings suggest that an enhanced hedonic response to palatable foods is associated with a loss of appetite control and constitute risk factors for overeating. This may contribute to the theoretical framework for understanding overconsumption in relation to weight gain.

### **10.3.3 Treatment and prevention**

While the current thesis examined the tendency to binge eat as a non-clinical occurrence the findings do highlight some possible targets for the treatment and prevention of binge eating tendencies. The finding that higher levels of binge eating tendencies were associated with higher levels of fat mass in both lean (Chapter 6) and overweight or obese individuals (Chapter 7 and 8) raises the question as to whether the greater levels of fat mass are a consequence or a cause of binge eating behaviour. The studies in the current thesis cannot determine causality with any certainty however prospective research in children and adolescents would allow us to better understand this association. Nevertheless, it can be suggested that treatment strategies should implement measures that target both the increased levels of fat mass and the greater binge eating tendencies in these individuals.

A fascinating anecdotal outcome emerged during the dietary recall procedure in Chapter 8 in which three participants, who were later identified as being in the O-B group, expressed surprise at the amount of sweet foods they had consumed over the 24-hour recall period. Indeed, the intake of sweet foods throughout the 24-hour recall period did not seem to be limited to one large eating occasion but were rather consumed across the day as snacks, and within meals. While further evidence is needed to corroborate these findings, a mindfulness based intervention may help to identify the causes of (e.g. low mood or stress, availability), and target these unhealthy eating habits by making individuals who score high in trait binge eating explicitly aware of them or the situations in which they are most vulnerable. Further to this, the consistent finding that O-B had greater implicit wanting for high-fat sweet foods when hungry, which may be a risk factor for overeating, suggests that avoidance of these types of food may be beneficial to increasing the control of appetite.

In a recent review article, Davis (2013) explores the notion of the progression of eating behaviour that may initially represent a form of passive overeating but then develop along a continuum into more severe and compulsive forms of overeating. Consistent with this, Stice et al., (2013) reported that subclinical levels of binge eating were associated with an enhanced risk of progression to BED over a period of eight years in a sample of female adolescents. In the current thesis, the finding from Chapter 8 that a small subset of O-B who met the criteria defined by the YFAS for food addiction also exhibited higher levels of eating pathology, may represent a progression of binge eating severity in line with the notion that Davis (2013) proposes. Long-term studies in either adults or in children and adolescents would therefore be beneficial to determine risk factors for such a progression and to identify targets for treatment and prevention of binge eating tendencies.

## **10.4 Limitations**

### **10.4.1 The Leeds Food Preference Questionnaire**

While the Leeds Food Preference Questionnaire has many advantages as a measure of liking and wanting for food, and has proven to be a reliable and versatile measure in this thesis there are some limitations that need to be considered with regards to its measurement of liking and wanting.

#### **10.4.1.1 Explicit liking**

Previous research has suggested that any subjective assessment of liking for food made in the absence of food consumption will rely on non-experiential sources of information such as memory and previous knowledge of the food (Robinson, Blissett, & Higgs, 2012; Robinson & Clore, 2002), which may result in the over-, or underestimation of liking for food. For example, Robinson et al. (2012) demonstrated that a recent disappointing food tasting experience impacted the rating of expected liking for food. However, compared to the relatively long-lasting effect on infrequently consumed foods, the effect on frequently consumed foods was short-lived. Indeed, a strength of the LFPQ's measure of liking is that the food images can be tailored so that participants' prior experience with and liking for the foods can be taken into consideration.

Further to this, the use of self-report techniques is vulnerable to the impact of social desirability and self-presentation biases, especially among individuals who are sensitive about their eating behaviour (Tooze et al., 2004). However, the present thesis has demonstrated that subjective ratings of liking for food are able to distinguish between overweight or obese individuals categorised as either binge-type or non-binge type, and between a fasted and fed state, suggesting that when used carefully self-report techniques can be sensitive to individual differences and motivational state.



#### **10.4.1.2 Implicit wanting**

In a review of the liking and wanting literature, Havermans et al., (2012) criticised the LFPQ's measurement of implicit wanting for the use of the validated algorithm, developed by Greenwalk, Nosek and Banaji, (2003), to transform reaction time data from the forced choice procedure into a standardised 'd score' (D-RT). However, the D-RT was designed to improve the statistical reliability, and reduce the contamination that arises from the large degree of individual variability that commonly occurs in reaction time data; therefore its use is not limited to the Implicit Association Task. In support of this, a very recent development has been made with regards to the scoring of the implicit wanting trials in which an appeal bias score for high fat versus low fat food for can be created using a frequency weighted algorithm (FWA) which results in the food category score being influenced by both selection (positively contributing to the score) and non-selection (negatively contributing to the score). The FWA has been developed specifically for the LFPQ and promisingly it correlates highly with the D-RT, supporting the use of the D-RT for implicit wanting. A further benefit of the FWA is that it accounts for every trial in which a food category is present and eliminates the need to remove individuals who always avoid one category, which is a limitation of the current D-RT procedure. However, the FWA does not allow for the analysis of specific food category preferences and therefore, the research question being examined will determine which method of scoring is appropriate.

#### **10.5 Future directions**

The work in this thesis has formed the basis of a Biotechnology and Biological Research Council grant application to examine the epigenetic mechanisms underlying the homeostatic and the hedonic processes of appetite in the context of food choice. Epigenetic control is the way in which the genome interacts with and responds to the environment resulting in certain functional modifications in the genome that do not arise from changes in the nucleotide sequence, but do appear to

be passed from one generation to the next (Haggarty, 2012; Vucetic, Kimmel, Totoki, Hollenbeck, & Reyes, 2010). To this end, under certain conditions two individuals (including identical twins) may have identical alleles but exhibit different phenotypes. Evidence from animal research suggests that epigenetic states in the brain associated with behaviours such as food choice are influenced by prenatal nutrition, in combination with emotional and psychological state, at key stages of development (Lillicrop, Phillips, Jackson, Hanson, & Burdge, 2005; Meaney & Szyf, 2005; Vucetic et al., 2010). To date, the epigenetic control of the individual behaviours and preferences that underlie phenotypes susceptible to overeating has not been examined. Using the binge-eating phenotype outlined in this thesis, the project aims to examine the epigenetic markers (linked, in a large population based study, to appetite and reward-based determinants of food choice) underlying the behaviours and processes that are associated with unhealthy food choice and susceptibility to overeating. Further to this, I will continue my work on susceptibility to overeating and hedonics by developing the concept of high-risk versus protective foods in a EUFP7 funded project – the SATiety Innovation Project (SATIN). SATIN will examine how the reformulation of foods can be used to help individuals who are susceptible to overeating and my part in the project will be to assess the acute effects of novel dietary components on liking and wanting in relation to satiation, satiety, and appetite-related peptides.

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## Appendices

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### Appendix 1

#### **1.1 Outcome of the principal components analysis on the COEQ.**

The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis as good, KMO = .788, and all KMO values for individual items were  $>.57$  which is within the acceptable limit. Bartlett's test of sphericity  $X^2(190) = 947.56$ ,  $p < 0.001$ , indicated that the correlations between the items were large enough for a PCA to be conducted. An initial analysis was conducted in order to obtain eigenvalues for each component in the data set. There were five components with an eigenvalue above 1 and in combination these components explained 59.92% of the variance. All five components were retained in the final analysis (see Table A1.1 for the factor loadings).

The items that cluster on the same components suggest that component 1 represents craving intensity, component 2 mood, component 3 craving for savoury foods, component 4 craving for sweet foods and component 5 feelings of fullness. The mean for each subscale will be calculated, and the number of items in the scale will divide this mean in order to obtain a subscale score. For the mood subscale, scores from the "How anxious have you felt item?" will be reversed.

Table A1.1 Pattern matrix for factor loadings following oblique rotation

	Component				
	1	2	3	4	5
How difficult has it been to resist any food cravings?	.84				
How often have you eaten in response to food cravings?	.80				
How strong have any food cravings been?	.74				
During the last 7 days how often have you had food cravings?	.65				
Generally, how difficult has it been to control your eating?	.63				
How hungry have you felt?					
How happy have you felt?		.89			
How anxious have you felt?		.86			
How alert have you felt?		.64			
How contented have you felt?		-.60			
How strong was your desire to eat savoury foods?			.74		
How often have you had cravings for starchy foods (bread, pasta)?			.73		
How often have you had cravings for dairy foods (cheese, yoghurt)?			.67		
How often have you had cravings for savoury foods (fries, crisps, burgers etc)?			.56		
How strong was your desire to eat sweet foods?				.72	
How often have you had cravings for chocolate and chocolate flavoured foods?				.66	
How often have you had cravings for fruit or fruit juice?				.58	
How often have you had cravings for other sweet foods (cakes, pastries, biscuits, etc)?				.56	
How difficult has it been to resist (problem food) during the last 7 days?				.54	
How full have you felt?					.88

## 2.1 Nutritional information for the food images in LFPQ and VPT

Table A2.1 Nutritional information and perceived qualities of standard set of food items used in the Leeds Food Preference Questionnaire and the Visual Probe Task.

HFSA	KCAL/100g	FAT/100g	PRO/100g	CHO/100g	FAT/kcal	PRO/kcal	CHO/kcal	%FAT	%PRO	%CHO	ED	Pleasant	Taste	Fat	Calories
Garlic bread	345	18.3	6.8	35.3	164.7	27.2	130.61	47.74	7.88	37.86	3.45	5.56	6.32	5.48	5.61
Crisps	537	34.1	5.9	49.7	306.9	23.6	183.89	57.15	4.39	34.24	5.37	5.52	6.19	5.93	5.68
Chips	239	12.4	3.2	30.5	111.6	12.8	112.85	46.69	5.36	47.22	2.39	5.61	5.88	6.24	6.01
Peanuts	590	49	27.6	10	441	110.4	37	74.75	18.71	6.27	5.9	5.29	5.85	5.83	5.42
Scotch egg*	235	14.3	10.6	16	128.7	42.4	59.2	54.77	18.04	25.19	2.35	4.11	6.05	5.66	5.53
LFSA	KCAL/100g	FAT/100g	PRO/100g	CHO/100g	FAT/kcal	PRO/kcal	CHO/kcal	%FAT	%PRO	%CHO	ED	Pleasant	Taste	Fat	Calories
Cucumber	21	0.3	0.8	3	2.7	3.2	11.10	12.86	15.24	52.86	0.21	4.48	5.44	1.16	1.25
Bread roll	245	3.4	11.3	39	30.6	45.2	144.30	12.49	18.45	58.90	2.45	5.09	5.3	3.27	3.64
Pilau rice	140	2.6	3	25.7	23.4	12	95.09	16.71	8.57	67.92	1.40	4.93	5.51	1.34	1.49
Potatoes	79	0.2	2.1	17.2	1.8	8.4	63.64	2.28	10.63	80.56	0.79	5.14	5.58	3.16	4.53
Peppers*	32	0.4	1	6.4	3.6	4	23.68	11.25	12.5	74	0.32	5.14	5.01	1.23	1.43
HFSW	KCAL/100g	FAT/100g	PRO/100g	CHO/100g	FAT/kcal	PRO/kcal	CHO/kcal	%FAT	%PRO	%CHO	ED	Pleasant	Taste	Fat	Calories
Jam biscuits	440	15	5.1	71.3	135	20.4	263.81	30.68	4.64	59.96	4.4	4.84	1.83	5.31	5.71
Doughnuts	410	21.2	6.9	48	190.8	27.6	177.60	46.54	6.73	43.32	4.1	5.39	1.52	6.52	6.64
Chocolate fingers	575	27	6.8	60.9	243	27.2	225.33	42.26	4.73	39.19	5.75	6.05	1.62	5.73	6.04
Chocolate	525	29.8	7.5	57	268.2	30	210.90	51.09	5.71	40.17	5.25	6.62	1.24	6.23	6.39
M&M's*	516	26.8	9.8	59	241.2	39.2	218.3	46.74	7.60	42.31	5.16	4.8	1.76	5.46	5.8
LFSW	KCAL/100g	FAT/100g	PRO/100g	CHO/100g	FAT/kcal	PRO/kcal	CHO/kcal	%FAT	%PRO	%CHO	ED	Pleasant	Taste	Fat	Calories
Apple	53	0.1	0.4	11.8	0.9	1.6	43.66	1.70	3.02	82.38	0.53	5.24	2.28	1.34	1.85
Strawberries	30	0.1	0.8	6	0.9	3.2	22.20	3.00	10.67	74.00	0.3	6.07	1.6	1.55	2.22
Skittles	403	4.2	0	90.5	37.8	0	334.85	9.38	0.00	83.09	4.03	5.23	1.18	3.13	5.14
Marshmallows	498	0	4	96.8	0	16	358.16	0.00	3.21	71.92	4.98	4.26	1.64	3.23	5.11
Fruit Salad*	43	0.2	0.6	9	1.8	2.4	33.3	4.19	5.58	77.44	0.43	5.87	1.83	1.43	1.94

*Note:* Items marked with an asterisk were used in the Visual Probe task only.



## Appendix 3

### 3.1 Food stimuli and non-food stimuli used in the Visual Probe Task

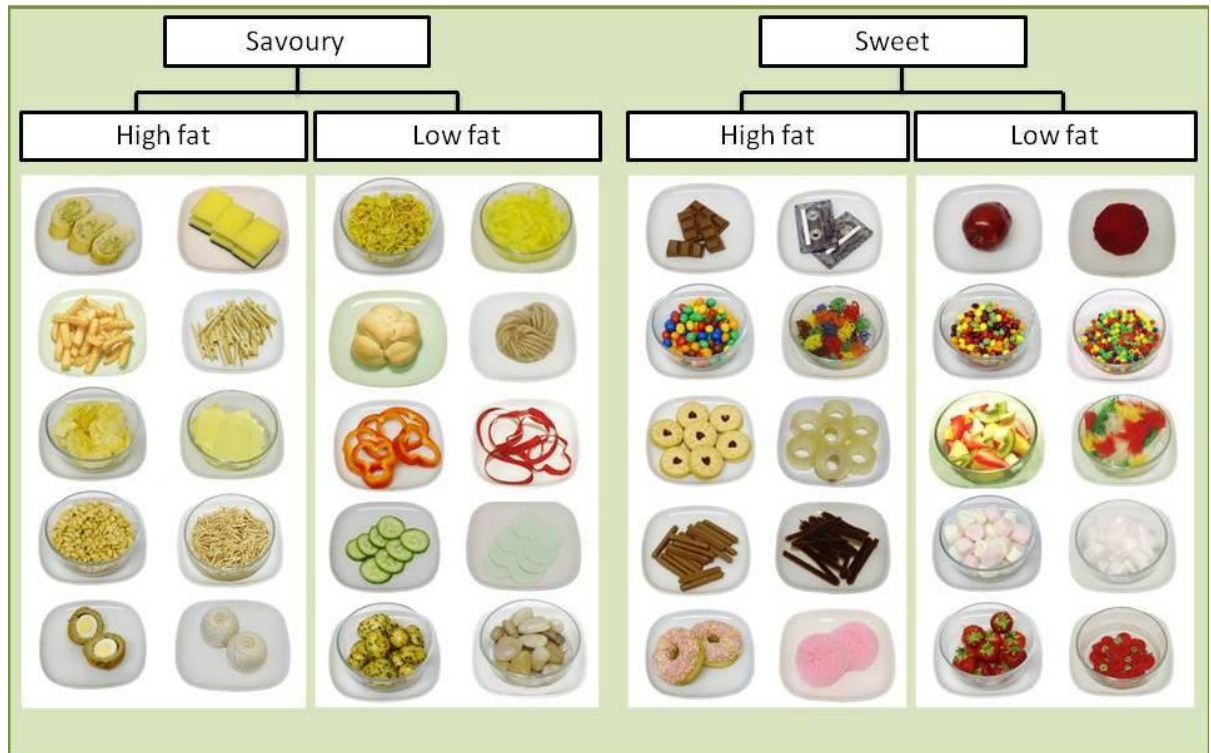


Figure A3.1 Food stimuli and their matched non-food controls used in the Visual Probe Task.

## Appendix 4

### 4.1 Nutritional information for the ad libitum snack task



Figure A4.1 Photograph of the presentation of snack foods used in the ad libitum eating task.

Table A4.1 Nutritional information for the snack food items used in the ad libitum eating task

	KCAL/100g	PRO/100g	CHO/100g	FAT/100g
Milk chocolate	530	7.5	57.0	29.8
Chocolate fingers	515	6.9	60.1	27.2
Cookies	484	5.6	64.2	22.1
Ready Salted crisps	526	6.1	51.5	31.9
Tortilla chips	506	7.4	57.9	26.5
Salted peanuts	609	26.3	6.9	24.2

## Appendix 5

### 5.1 Pearson's correlations between 100ms and 500ms attentional bias and explicit liking and wanting

Table A5.1 Pearson's correlations between 100ms and 500ms attentional bias scores and explicit liking in the fasted and fed condition

		100ms				500ms			
Fasted		HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Explicit liking	HFSA	.006	.073	-.054	.252	-.063	.209	-.184	.066
	LFSA	.088	.019	.198	.166	-.054	.083	-.154	.033
	HFSW	-.118	.013	.126	.075	.073	.181	.095	-.067
	LFSW	-.130	-.203	.138	.040	-.015	-.144	.149	-.190
Fed		HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Explicit liking	HFSA	.150	.030	.257	.107	-.175	-.179	-.176	.120
	LFSA	.183	.144	-.018	.193	-.219	-.180	.047	.094
	HFSW	-.035	-.018	.142	-.021	.135	.007	.047	.190
	LFSW	.069	.142	-.025	.146	.099	.035	.134	.235

Table A5.2 Correlational analysis between 100ms and 500ms attentional bias scores and explicit wanting in the fasted and fed condition

		100ms				500ms			
Fasted		HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Explicit wanting	HFSA	-.034	.017	-.028	.212	-.098	.169	-.200	.122
	LFSA	-.041	-.069	.212	.119	-.119	.048	-.154	-.001
	HFSW	-.177	-.001	.002	.049	.041	.159	.080	.073
	LFSW	-.046	-.163	.136	.058	-.060	-.165	.169	-.106
Fed		HFSA	LFSA	HFSW	LFSW	HFSA	LFSA	HFSW	LFSW
Explicit wanting	HFSA	.127	.011	.192	.083	-.182	-.184	-.140	.142
	LFSA	.174	.130	.060	.207	-.158	-.101	.001	.156
	HFSW	-.087	-.027	.122	.005	.185	.051	.007	.177
	LFSW	.070	.143	-.028	.245	.073	-.038	.123	.267

## Appendix 6

### **6.1 Outcome of the ANCOVA to examine the influence of trait binge eating on 100ms and 500ms attentional bias**

#### **100ms trials**

There was no main effect of condition [ $F(1, 51) = 2.11, p > 0.05$ ] or category [ $F(3, 153) = 1.43, p > 0.05$ ]. There were no interactions between BES and category [ $F(3, 153) = 2.27, p > 0.05$ ] or between BES and condition [ $F(1, 51) = 2.82, p > 0.05$ ]. Finally there was no interaction between condition, category and BES [ $F(3, 153) = .316, p > 0.05$ ].

#### **500ms trials**

There was no main effect of condition [ $F(1, 51) = .248, p > 0.05$ ] or category [ $F(3, 153) = .142, p > 0.05$ ]. There were no interactions between BES and category [ $F(3, 153) = .159, p > 0.05$ ] or between BES and condition [ $F(1, 51) = .921, p > 0.05$ ]. Finally there was no interaction between condition, category and BES [ $F(3, 153) = .218, p > 0.05$ ].

## Appendix 7

### 7.1 Example 24-hour dietary recalls for O-B

Table A7.1 24-hour dietary recall for an O-B with a BES score of 18

BMI: 27.7 Binge eating score: 18 Estimated daily energy requirements: 2678.90 calories TM-EI: 3357.44 calories DR-EI: 2434.23 calories		
Breakfast	Portion	Calories
<i>Sainsbury's</i> Porridge oats	50g	180
<i>Alpro</i> Soya milk	300ml	132
Granulated sugar	30g	120
Banana	x1	96.12
Morning snacks		
None reported.		
Lunch		
<i>Tesco</i> Egg mayonnaise sandwich	x1	495.07
<i>McCoy's</i> Prawn crisps	32g	168.32
Coca-cola	330ml	138.60
Afternoon snacks		
<i>Galaxy</i> Caramel	50g	249
Dinner		
Boiled egg	x2	184.8
<i>Hovis</i> White bread – toasted	94g	239
<i>Tesco</i> Spread	15g	80.40
Evening snacks		
<i>Galaxy</i> Caramel	50g	249
<i>Morrisons</i> Toffee Pecan ice-cream	98ml	101.92

Table A7.2 24-hour dietary recall for an O-B with a BES score of 21

BMI:	27.7	
Binge eating score:	21	
Estimated daily energy requirements:	2505.70 calories	
TM-EI:	3182.31 calories	
DR-EI:	3662.74 calories	
Breakfast	Portion	Calories
<i>Galaxy Orange &amp; shortcake bar</i>	40g	217.6
<i>Sainsbury's Large white chocolate chip cookies</i>	x2	484
Morning snacks		
None reported.		
Lunch		
<i>Greggs Steak bake</i>	272g	859.98
<i>Dr. Pepper Zero</i>	500ml	5
<i>Greggs Iced bun</i>	37g	190
Afternoon snacks		
<i>Cadbury's bar and a half</i>	75g	416.25
Dinner		
<i>Sainsbury's Mixed vegetable pack</i>	240g	67.2
<i>Sainbury's Medium grated cheese</i>	36g	140.04
<i>Kewpie Mayonnaise</i>	30g	214.29
<i>Sainsbury's Baked beans</i>	420g	340.20
<i>Sainbury's Medium grated cheese</i>	48g	186.72
<i>Ben &amp; Jerry's Cookie dough ice-cream</i>	125ml	287.50
Evening snacks		
<i>Asda White bread – toasted</i>	92g	218.96
<i>Sainsbury's Light spread</i>	10g	35

Table A7.3 24-hour dietary recall for an O-B with a BES score of 25

BMI:	28.2	
Binge eating score:	25	
Estimated daily energy requirements:	2712.20 calories	
TM-EI:	4037.17 calories	
DR-EI:	3650.92 calories	
Breakfast	Portion	Calories
<i>Tesco</i> Cornflakes	50g	190
<i>Kellogg's</i> Branflakes	20g	66.8
<i>Tesco</i> Semi-skimmed milk	300ml	147
Morning snacks		
<i>Pret a manger</i> Almond croissant	x1	365
Lunch		
<i>Covent Garden</i> Spiced carrot soup	250g	90
<i>Pret a manger</i> White chocolate & raspberry cookie	x1	356
Afternoon snacks		
<i>Mr. Kipling</i> Bakewell tart (large pie)	276g	1150.92
Dinner		
Spaghetti	158g	282.03
<i>Quorn</i> Soya mince	100g	100
Yellow onion	60g	23.4
Spring onion	50g	12.5
Olive oil	30ml	270
<i>Dolmio</i> Pasta sauce	167g	68.33
<i>Tesco</i> Grated cheese	40g	155.60
Evening snacks		
<i>Hovis</i> Seeded sensations bread – toasted	88g	244.64
<i>Tesco</i> Light spread	15g	52.5
<i>Tesco</i> Strawberry jam	30g	76.2

## **Appendix 8**

### **8.1 Debriefing statement for the study presented in Chapter 7**

Thank you for your time and participation in the current study – it is greatly appreciated.

The purpose of the study was to examine individual differences in responsiveness to food cues during two different motivational states – hungry and fed.

Hunger appears to be a powerful motivational force that is able to alter aspects of cognition (Piech et al., in press), and enhance motivation toward food cues and food (Castellanos et al., 2009). We aimed to assess whether motivational responses to food, as measured by the Leeds Food Preference Questionnaire (LFPQ, Finlayson et al. 2008), would be different under states of hunger and satiation and how these states impact snacking behaviour. In addition to participating in the test sessions you completed a number of questionnaires, these assess various eating behaviour traits. We will be using these traits to investigate individual differences in the motivational response to food and its cues.

If you have any additional questions regarding this research please feel free to ask the experimenter now.

Once again, thank you for your participation.



## Appendix 9

### 9.1 Summary of inferential statistics for Chapter 9

Table A9.1 Summary table of inferential statistics for reward-related genes and energy intake

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
OPRM1	rs1799971	Overall energy intake	1, 168	.053	0.82
		Sweet energy intake	1, 168	.714	0.39
		Savoury energy intake	1, 168	.492	0.48
	rs495491	Overall energy intake	1, 157	.187	0.67
		Sweet energy intake	1, 157	.465	0.49
		Savoury energy intake	1, 157	.015	0.90
DRD2	rs6277	Overall energy intake	2, 157	4.16	<b>0.02</b>
		Sweet energy intake	2, 157	5.73	<b>0.01</b>
		Savoury energy intake	2, 157	.372	0.69
ANKK1	rs1800497	Overall energy intake	1, 157	.165	0.69
		Sweet energy intake	1, 157	.000	0.99
		Savoury energy intake	1, 157	.486	0.48

Table A9.2 Summary table of inferential statistics for taste-related genes and energy intake

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
CD36	rs2151916	Overall energy intake	2, 155	.764	0.46
		Sweet energy intake	2, 155	.783	0.46
		Savoury energy intake	2, 155	.143	0.87
	rs1761667	Overall energy intake	2, 155	.672	0.51
		Sweet energy intake	2, 155	.271	0.76
		Savoury energy intake	2, 155	.573	0.57
TAS1R2	rs35874116	Overall energy intake	1, 159	.358	0.55
		Sweet energy intake	1, 159	.261	0.61
		Savoury energy intake	1, 159	.149	0.70
TAS2R38	rs1726866	Overall energy intake	2, 141	1.14	0.32
		Sweet energy intake	2, 141	.291	0.75

SLC2A2	rs5400	Savoury energy intake	2, 141	3.17	<b>0.05</b>
		Overall energy intake	1, 157	1.42	0.24
		Sweet energy intake	1, 157	.000	0.98
		Savoury energy intake	1, 157	4.49	<b>0.04</b>

Table A9.3 Summary table of inferential statistics for obesity-related genes and energy intake

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
FTO	rs9939609	Overall energy intake	2, 151	2.80	0.06
		Sweet energy intake	2, 151	5.77	<b>0.01</b>
		Savoury energy intake	2, 151	.028	0.97
	rs1121980	Overall energy intake	2, 156	3.54	<b>0.03</b>
		Sweet energy intake	2, 156	5.66	<b>0.01</b>
		Savoury energy intake	2, 156	.196	0.82
MC4R	rs17782313	Overall energy intake	1, 157	.513	0.48
		Sweet energy intake	1, 157	.002	0.96
		Savoury energy intake	1, 157	1.39	0.24

Table A9.4 Summary table of inferential statistics for reward-related genes and anthropometrics and body composition

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
OPRM1	rs1799971	BMI (kg/m <sup>2</sup> )	1, 172	2.48	0.12
		Fat mass (kg)	1, 159	0.15	0.90
		Fat free mass (kg)	1, 161	.667	0.42
		Body fat (%)	1, 159	.305	0.58
		Waist (cm)	1, 169	1.66	0.20
	rs495491	BMI (kg/m <sup>2</sup> )	1, 162	.105	0.75
		Fat mass (kg)	1, 153	.184	0.67
		Fat free mass (kg)	1, 155	.264	0.61
		Body fat (%)	1, 153	.342	0.56
		Waist (cm)	1, 159	.018	0.89
	DRD2	BMI (kg/m <sup>2</sup> )	2, 160	5.04	<b>0.01</b>
		Fat mass (kg)	2, 151	1.89	0.16
		Fat free mass (kg)	2, 153	.814	0.45
		Body fat (%)	2, 151	2.11	0.13
	ANKK1	Waist (cm)	2, 157	3.34	<b>0.04</b>
		BMI (kg/m <sup>2</sup> )	1, 162	4.41	<b>0.05</b>
		Fat mass (kg)	1, 153	1.95	0.16
		Fat free mass (kg)	1, 155	1.20	0.28

Body fat (%)	1, 153	2.66	0.11
Waist (cm)	1, 159	2.40	0.12

Table A9.5 Summary table of inferential statistics for taste-related genes and anthropometrics and body composition

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
CD36	rs2151916	BMI (kg/m <sup>2</sup> )	2, 160	2.29	0.11
		Fat mass (kg)	2, 151	2.41	0.09
		Fat free mass (kg)	2, 153	1.26	0.29
		Body fat (%)	2, 151	2.04	0.13
		Waist (cm)	2, 158	3.84	<b>0.02</b>
	rs1761667	BMI (kg/m <sup>2</sup> )	2, 160	.071	0.93
		Fat mass (kg)	2, 151	.271	0.76
		Fat free mass (kg)	2, 153	.628	0.54
		Body fat (%)	2, 151	.231	0.79
		Waist (cm)	2, 157	.860	0.43
TAS1R2	rs35874116	BMI (kg/m <sup>2</sup> )	1, 164	8.14	<b>0.01</b>
		Fat mass (kg)	1, 155	6.13	<b>0.02</b>
		Fat free mass (kg)	1, 155	1.90	0.17
		Body fat (%)	1, 155	5.64	<b>0.02</b>
		Waist (cm)	1, 161	5.78	<b>0.02</b>
TAS2R38	rs1726866	BMI (kg/m <sup>2</sup> )	2, 144	1.68	0.19
		Fat mass (kg)	2, 135	2.21	0.12
		Fat free mass (kg)	2, 137	2.35	0.10
		Body fat (%)	2, 135	1.36	0.26
		Waist (cm)	2, 142	1.24	0.29
SLC2A2	rs5400	BMI (kg/m <sup>2</sup> )	1, 162	.400	0.52
		Fat mass (kg)	1, 154	.323	0.58
		Fat free mass (kg)	1, 156	.408	0.52
		Body fat (%)	1, 154	.022	0.88
		Waist (cm)	1, 159	.091	0.76

Table A9.6 Summary table of inferential statistics for obesity-related genes and anthropometrics and body composition

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
FTO	rs9939609	BMI (kg/m <sup>2</sup> )	2, 156	1.36	0.26
		Fat mass (kg)	2, 148	1.74	0.18
		Fat free mass (kg)	2, 150	1.94	0.15
		Body fat (%)	2, 148	1.66	0.19
		Waist (cm)	2, 153	.325	0.72
	rs1221980	BMI (kg/m <sup>2</sup> )	2, 161	.922	0.40
		Fat mass (kg)	2, 152	1.28	0.28
		Fat free mass (kg)	2, 154	1.66	0.19
		Body fat (%)	2, 152	1.23	0.29
		Waist (cm)	2, 158	.297	0.74
MC4R	rs17782313	BMI (kg/m <sup>2</sup> )	1, 162	.493	0.48
		Fat mass (kg)	1, 153	1.98	0.16
		Fat free mass (kg)	1, 155	.492	0.48
		Body fat (%)	1, 153	1.55	0.22

Waist (cm)	1, 159	1.59	0.21
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Table A9.7 Summary table of inferential statistics for reward-related genes and food hedonics

Gene	SNP	Dependent variable	Factor	Degrees of freedom	F value	p value
OPRM1	rs1799971	Explicit liking	Fat x G	1, 166	.017	0.89
			Taste x G	1, 166	.351	0.55
			Fat x Taste x G	1, 166	.510	0.47
		Explicit wanting	Fat x G	1, 166	.439	0.51
			Taste x G	1, 166	.452	0.50
			Fat x Taste x G	1, 166	.338	0.56
		Implicit wanting	Fat x G	1, 163	.053	0.82
			Taste x G	1, 163	.024	0.87
			Fat x Taste x G	1, 163	.343	0.55
	rs495491	Explicit liking	Fat x G	1, 156	.985	0.32
			Taste x G	1, 156	2.93	0.09
			Fat x Taste x G	1, 156	.501	0.48
		Explicit wanting	Fat x G	1, 156	.991	0.32
			Taste x G	1, 156	1.93	0.17
			Fat x Taste x G	1, 156	.160	0.69
		Implicit wanting	Fat x G	1, 153	1.73	0.19
			Taste x G	1, 153	2.47	0.12
			Fat x Taste x G	1, 153	.538	0.46
DRD2	rs6277	Explicit liking	Fat x G	2, 154	2.46	0.09
			Taste x G	2, 154	2.82	0.08
			Fat x Taste x G	2, 154	.055	0.95
		Explicit wanting	Fat x G	2, 154	2.14	0.12
			Taste x G	2, 154	.017	0.89
			Fat x Taste x G	2, 154	.023	0.97
		Implicit wanting	Fat x G	2, 151	.965	0.38
			Taste x G	2, 151	1.09	0.29
			Fat x Taste x G	2, 151	1.28	0.28
ANKK1	rs1800497	Explicit liking	Fat x G	1, 157	1.05	0.31
			Taste x G	1, 157	.041	0.84
			Fat x Taste x G	1, 157	1.79	0.18
		Explicit wanting	Fat x G	1, 157	.833	0.36
			Taste x G	1, 157	.357	0.55
			Fat x Taste x G	1, 157	1.21	0.27
		Implicit wanting	Fat x G	1, 154	.213	0.65
			Taste x G	1, 154	.012	0.91
			Fat x Taste x G	1, 154	1.91	0.16

Table A9.8 Summary table of inferential statistics for taste-related genes and food hedonics

Gene	SNP	Dependent variable	Factor	Degrees of freedom	F value	p value
CD36	rs2151916	Explicit liking	Fat x G	2, 154	.353	0.70
			Taste x G	2, 154	1.03	0.36
			Fat x Taste x G	2, 154	.152	0.86

TAS1R2	rs1761667	Explicit wanting	Fat x G	2, 154	.207	0.81
			Taste x G	2, 154	.611	0.54
			Fat x Taste x G	2, 154	.424	0.66
		Implicit wanting	Fat x G	2, 151	2.14	0.12
			Taste x G	2, 151	1.32	0.27
			Fat x Taste x G	2, 151	.086	0.92
		Explicit liking	Fat x G	2, 154	1.21	0.30
			Taste x G	2, 154	.291	0.75
			Fat x Taste x G	2, 154	1.19	0.31
		Explicit wanting	Fat x G	2, 154	.807	0.45
			Taste x G	2, 154	.165	0.85
			Fat x Taste x G	2, 154	.169	0.85
	rs35874116	Implicit wanting	Fat x G	2, 151	.544	0.58
			Taste x G	2, 151	.409	0.67
			Fat x Taste x G	2, 151	.398	0.67
		Explicit liking	Fat x G	2, 158	.244	0.79
			Taste x G	2, 158	1.01	0.37
			Fat x Taste x G	2, 158	.695	0.50
		Explicit wanting	Fat x G	2, 158	.294	0.75
			Taste x G	2, 158	.509	0.60
			Fat x Taste x G	2, 158	.496	0.61
		Implicit wanting	Fat x G	2, 155	.281	0.76
			Taste x G	2, 155	.486	0.61
			Fat x Taste x G	2, 155	.581	0.56
TAS2R38	rs1726866	Explicit liking	Fat x G	2, 139	.411	0.66
			Taste x G	2, 139	.721	0.48
			Fat x Taste x G	2, 139	.909	0.41
		Explicit wanting	Fat x G	2, 139	.232	0.79
			Taste x G	2, 139	.273	0.76
			Fat x Taste x G	2, 139	.813	0.45
		Implicit wanting	Fat x G	2, 136	.407	0.66
			Taste x G	2, 136	1.70	0.19
			Fat x Taste x G	2, 136	1.61	0.21
	rs5400	Explicit liking	Fat x G	1, 156	.249	0.62
			Taste x G	1, 156	3.24	<b>0.07</b>
			Fat x Taste x G	1, 156	1.22	0.27
		Explicit wanting	Fat x G	1, 156	.205	0.65
			Taste x G	1, 156	2.36	0.13
			Fat x Taste x G	1, 156	1.66	0.20
		Implicit wanting	Fat x G	1, 153	.040	0.84
			Taste x G	1, 153	.371	0.54
			Fat x Taste x G	1, 153	2.72	0.10

Table A9.9 Summary table of inferential statistics for obesity-related genes and food hedonics

Gene	SNP	Dependent variable	Factor	Degrees of freedom	F value	p value
FTO	rs9939609	Explicit liking	Fat x G	2, 151	1.78	0.17
			Taste x G	2, 151	.558	0.57
			Fat x Taste x G	2, 151	.141	0.87
		Explicit wanting	Fat x G	2, 151	2.36	0.09
			Taste x G	2, 151	.457	0.63
			Fat x Taste x G	2, 151	.520	0.59
		Implicit wanting	Fat x G	2, 148	.715	0.49

MC4R	rs1121980	Explicit liking	Taste x G	2, 148	1.20	0.30
			Fat x Taste x G	2, 148	.682	0.51
			Fat x G	2, 156	1.52	0.22
		Explicit wanting	Taste x G	2, 156	.654	0.52
			Fat x Taste x G	2, 156	.125	0.88
			Fat x G	2, 156	2.24	0.11
		Implicit wanting	Taste x G	2, 156	.567	0.57
			Fat x Taste x G	2, 156	.416	0.66
			Fat x G	2, 153	.224	0.80
	rs17782313	Explicit liking	Taste x G	2, 153	1.03	0.36
			Fat x Taste x G	2, 153	.722	0.49
			Fat x G	1, 156	.018	0.89
		Explicit wanting	Taste x G	1, 156	2.57	0.11
			Fat x Taste x G	1, 156	3.38	0.07
			Fat x G	1, 156	.544	0.46
		Implicit wanting	Taste x G	1, 156	.225	0.64
			Fat x Taste x G	1, 156	.544	0.46
			Fat x G	1, 153	.001	0.98
	Taste x G	1, 153	1.12	0.29		
	Fat x Taste x G	1, 153	1.18	0.28		

Table A9.10 Summary table of inferential statistics for reward-related genes and psychometric traits

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
OPRM1	rs1799971	Restraint	1, 165	.119	0.73
		Disinhibition	1, 168	.010	0.92
		Hunger	1, 167	1.98	0.16
		BAS D	1, 172	.068	0.79
		BAS FS	1, 170	.166	0.68
		BAS RR	1, 171	1.65	0.20
		BES	1, 167	.855	0.36
	rs495491	Restraint	1, 154	.090	0.76
		Disinhibition	1, 158	.112	0.74
		Hunger	1, 157	.587	0.45
		BAS D	1, 162	.922	0.34
		BAS FS	1, 160	.536	0.47
		BAS RR	1, 160	.073	0.79
		BES	1, 157	.188	0.67
DRD2	rs6277	Restraint	2, 153	.064	0.94
		Disinhibition	2, 156	.106	0.90
		Hunger	2, 155	.219	0.80
		BAS D	2, 160	.156	0.86
		BAS FS	2, 158	.435	0.65
		BAS RR	2, 158	.919	0.40
		BES	2, 155	.468	0.63
ANKK1	rs1800497	Restraint	1, 154	1.75	0.19
		Disinhibition	1, 158	1.96	0.16
		Hunger	1, 157	1.17	0.28
		BAS D	1, 162	2.52	0.11
		BAS FS	1, 160	5.25	<b>0.02</b>

BAS RR	1, 160	5.15	<b>0.03</b>
BES	1, 157	1.65	0.20

Table A9.11 Summary table of inferential statistics for taste-related genes and psychometric traits

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
CD36	rs2151916	Restraint	2, 152	2.08	0.13
		Disinhibition	2, 156	2.17	0.12
		Hunger	2, 155	.570	0.57
		BAS D	2, 160	.425	0.65
		BAS FS	2, 158	.028	0.97
		BAS RR	2, 158	.065	0.94
		BES	2, 155	1.13	0.33
		Restraint	2, 153	1.95	0.15
	rs1761667	Disinhibition	2, 156	.858	0.43
		Hunger	2, 155	1.05	0.35
		BAS D	2, 160	1.45	0.24
		BAS FS	2, 158	.085	0.92
		BAS RR	2, 158	1.41	0.25
		BES	2, 155	.448	0.64
TAS1R2	rs35874116	Restraint	1, 156	.065	0.80
		Disinhibition	1, 160	.546	0.46
		Hunger	1, 159	.354	0.55
		BAS D	1, 164	.036	0.85
		BAS FS	1, 162	.024	0.88
		BAS RR	1, 162	.000	0.99
		BES	1, 159	2.61	0.11
TAS2R38	rs1726866	Restraint	2, 139	3.47	<b>0.03</b>
		Disinhibition	2, 141	.628	0.54
		Hunger	2, 140	1.26	0.29
		BAS D	2, 144	.034	0.96
		BAS FS	2, 143	.362	0.70
		BAS RR	2, 144	1.22	0.29
		BES	2, 140	1.12	0.33
SLC2A2	rs5400	Restraint	1, 154	.651	0.42
		Disinhibition	1, 158	1.18	0.28
		Hunger	1, 157	.141	0.71
		BAS D	1, 163	.041	0.84
		BAS FS	1, 161	.381	0.54
		BAS RR	1, 161	2.44	0.12
		BES	1, 157	1.53	0.22

Table A9.12 Summary table of inferential statistics for obesity-related genes and psychometric traits

Gene	SNP	Dependent variable	Degrees of freedom	F value	p value
FTO	rs9939609	Restraint	2, 149	1.03	0.36
		Disinhibition	2, 152	.226	0.79
		Hunger	2, 151	.667	0.52
		BAS D	2, 157	.750	0.47
		BAS FS	2, 155	.835	0.44
		BAS RR	2, 155	.765	0.47
		BES	2, 151	.154	0.86
	rs1121980	Restraint	2, 154	1.49	0.23
		Disinhibition	2, 157	.022	0.98
		Hunger	2, 156	.241	0.79
		BAS D	2, 161	.549	0.57
		BAS FS	2, 159	.407	0.67
		BAS RR	2, 159	.857	0.43
		BES	2, 156	.108	0.89
MC4R	rs17782313	Restraint	1, 154	.192	0.66
		Disinhibition	1, 158	.011	0.92
		Hunger	1, 157	.251	0.62
		BAS D	1, 162	.547	0.45
		BAS FS	1, 160	.536	0.47
		BAS RR	1, 160	1.59	0.21
		BES	1, 157	.105	0.75



## Appendix 10

### 10.1 The effect of Taq1A genotype on implicit wanting for food in the Leeds sample

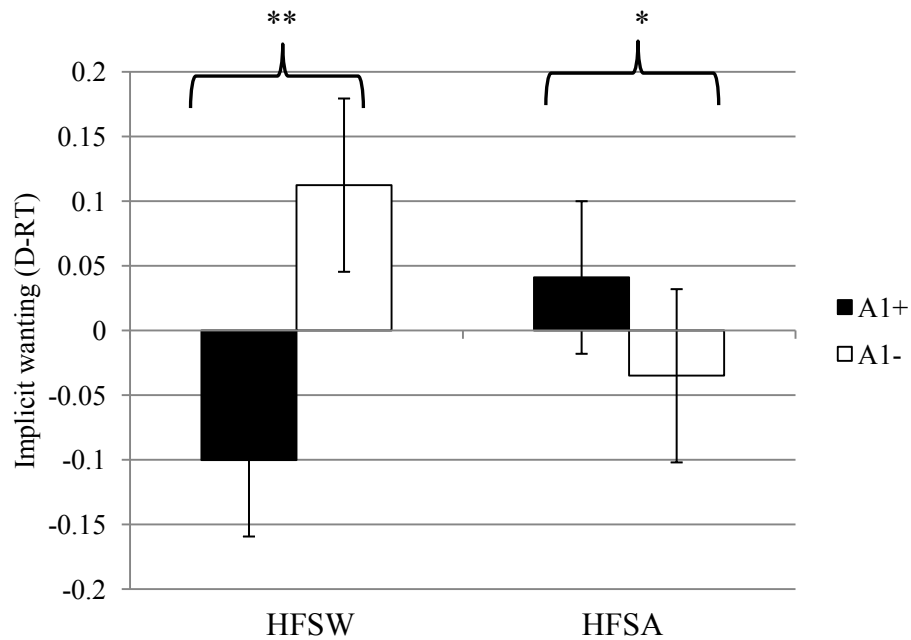


Figure A10.1 Implicit wanting (D-RT) for high-fat sweet and high-fat savoury according to ANKK1 rs1800497 genotype.

\* $p < 0.05$ ; \*\* $p < 0.01$

Figure A9.1 illustrates that A1- had significantly greater implicit wanting for HFSW [ $t(82) = 2.48$ ,  $p < 0.01$ ] compared to A1+. Whereas A1+ had significantly higher implicit wanting for HFSA [ $t(82) = 1.94$ ,  $p < 0.05$ ] compared to A1-.

## **Appendix 11**

### **11.1 Multiple regression analyses to examine the predictors of the behavioural tendencies in the trait binge eating phenotype**

In order to examine the psychometric predictors of preference for high-fat sweet foods, and implicit wanting for high-fat sweet foods, multiple regression analyses were conducted with trait binge eating, trait disinhibition and trait hunger entered as predictor variables. Multiple regression analyses were conducted for the studies presented in Chapter 7 and Chapter 8.

#### **11.1.1 Chapter 7**

##### **11.1.1.1 Energy intake**

The results of the regression indicated that when trait binge eating, trait disinhibition and trait hunger were entered together they explained 30% of the variance in energy intake from sweet foods under laboratory conditions [ $R^2 = .30$ ,  $F(3, 45) = 6.00$ ,  $p < 0.01$ ]. Trait binge eating was the only significant predictor accounting for 8% unique variance [ $t = 2.03$ ,  $\beta = .457$ ,  $p < 0.05$ ].

#### **11.1.2 Chapter 8**

##### **11.1.2.1 Energy intake**

The results of the regression indicated that when trait binge eating, trait disinhibition and trait hunger were entered together they explained 31% of the variance in energy intake from sweet foods under laboratory conditions [ $R^2 = .31$ ,  $F(3, 33) = 5.91$ ,  $p < 0.01$ ]. Trait binge eating was the only significant predictor accounting for 17% unique variance [ $t = 2.86$ ,  $\beta = .590$ ,  $p < 0.01$ ]. When the predictor variables were examined with regards to the number of sweet snack items consumed under free-living conditions, 38% of the variance was explained [ $R^2 = .38$ ,  $F(3, 33) = 6.19$ ,  $p < 0.01$ ]. Trait binge was the only significant predictor accounting for 29% unique variance [ $t = 3.73$ ,  $\beta = .769$ ,  $p < 0.001$ ].

### **11.1.2.2 Implicit wanting**

The results of the regression indicated that when trait binge eating, trait disinhibition and trait hunger were entered together they explained 41% of the variance in implicit wanting for high-fat sweet food [ $R^2 = .41$ ,  $F(3, 33) = 6.88$ ,  $p < 0.001$ ]. Trait binge eating was the only significant predictor accounting for 12% unique variance [ $t = 2.41$ ,  $\beta = .487$ ,  $p < 0.02$ ].

## **Appendix 12**

### **12.1 Hierarchical multiple regression to assess incremental validity of implicit wanting**

To assess the incremental validity of implicit wanting, two hierarchical multiple regression analyses were conducted with explicit liking and implicit wanting for high-fat sweet foods entered as predictor variables of overall energy intake. In step one explicit liking was entered alone, and in step two explicit liking was entered with implicit wanting. Explicit liking was a significant predictor of overall energy intake [ $t = 2.14$ ,  $\beta = .293$   $p < 0.05$ ] explaining 7% of the variance, when implicit wanting was entered it was also a significant predictor and accounted for 14.9% unique variance [ $t = 3.02$ ,  $\beta = .398$ ,  $p < 0.01$ ].